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ANTERIOR OPEN-BITE OR RETROGNATHIA CORRECTED BY A VERTICAL SLIDING OSTEOTOMY OF THE RAMUS, WITH OR WITHOUT BONE GRAFT: AN IMPROVED TECHNIQUE

Preliminary Report of Two Cases

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MANY surgical procedures have been recommended for correction of anterior open-bite and mandibular retrognathia. The following classification of retrognathia versus micrognathia has been discussed by Harry Sicher and myself and is presented to help standardize the nomenclature of this type of jaw deformity.

By retrognathia of the mandible we mean a mandible whose ramus is underdeveloped, so that the chin is in a retruded position although the mandibular dental arch may be of normal size.

Mandibular retrognathism (Class II malocclusion) is the counterpart of mandibular prognathism (Class III malocclusion). In retrognathism the mandible is too small for the rest of the skull, and in prognathism it is too large. Extreme variations in the size of the mandible (small or large) should be differentiated from these cases and designated as micrognathia and macrognathia.

As a rule, true micrognathia is the sequela of an early injury to the condyle, with resultant destruction of the cartilaginous growth center and frequently with partial or complete ankylosis. Micrognathia is characterized by the prominent antegonial notch and usually by an anterior open-bite.

As a rule, true macrognathia is a symptom of acromegaly and is characterized by the entire overgrowth of the mandible, causing the teeth to be separated.

TECHNIQUE

It is essential that a complete history, physical examination, and laboratory examination be completed first. The roentgenographic examination should consist of a centric profile view, as used in orthodontic surveys, a posteroanterior

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view of the mandible, oblique views of the rami of the mandible, and full-mouth periapical series. The oblique views of the rami should be taken so as to give the least distortion in the actual size of the ramus and to show the exact location of the mandibular nerve canal within the ramus. This will aid in establishing the exact course and position of the osteotomy and the position of the interosseous wire suture holes. Accurate study models of the dental arches should be mounted on the articulator in the exact preoperative position. Another set of study models should be mounted on an anatomic articulator in the desired postoperative position, with the second molar teeth kept separated between 1 and 2 mm. while the anterior bite is totally closed. The teeth should be placed arbitrarily into the best intermaxillary relationship. Consultation with an orthodontist or prosthodontist is advisable, especially if his services will be required following corrective surgery. An acrylic splint can be made of clear, fast-cure acrylic to just cover the occlusal edges and lateral margins of all the teeth. Preoperative profile and straight-on photographs are a useful part of the patient's record.

A normal orthodontic tracing can be made from the centric profile roentgenograms. By using this tracing, one can make a projected cut in the ramus, running from the posterior third of the sigmoid notch to just forward of the angle of the mandible and staying just posterior to the entrance of the mandibular nerve on the medial surface of the ramus. The oblique films will make it easier to determine how much space there is between the nerve and the posterior margin of the ramus. The osteotomy is run parallel to the posterior border of the ramus of the mandible. This distance is usually $\frac{5}{16}$ to $\frac{7}{16}$ inch forward of the posterior margin of the ramus. On the tracing, when this cut has been completed and the anterior section of the mandible has been cut out, it can then be moved into its corrected position and the new position of the forward section of the ramus can be accurately determined. From this tracing one can determine the amount of separation between the fragments and decide whether it is possible to obtain union by rotating the condylar fragment forward or whether a bone graft is needed to get union. The tracing may be drawn differently for the two sides if asymmetry is present. The acrylic splint should be fitted on the two separate arches to correct for interferences a few days before surgery.

One day before surgery fracture arch bars are placed on both the upper and the lower jaws and a circumferential 22 gauge wire is placed around the symphysis and the mandibular arch bar to hold the anterior segment of the bar in its proper position throughout the period of immobilization.

SURGERY

The curved skin incision is made 3 cm. in length and about 2 cm. below the angle of the mandible in a skin line, if one is present. The incision is carried down through the platysma, avoiding injury to the mandibular branch of the facial nerve. The external maxillary artery and vein, when identified, may be retracted forward. An incision is made through the periosteum and attachment fibers of the masseter muscle, which are elevated with a sharp

chisel or periosteotome, from the lateral surface of the ramus until the sigmoid notch is visualized. By means of a specially adapted nasal bone saw (Fig. 1), or a spiral cross-cut drill as recommended by Thoma,[2] the bone is cut from the sigmoid notch to forward of the angle of the mandible. This cut should run parallel to the posterior border of the ramus, but 1 to 3 mm. behind the mandibular nerve foramen located medially. The bone in this area is not too thick and is easily sectioned. If the cut in the middle of the ramus is not quite through the bone, then the bone can easily be separated by turning a periosteotome in the groove. This prevents the possible disturbance of the nerve and vessel in case it should be just under the bone incision. After division of the bone, the periosteum under the proximal and distal fragments can be elevated so that the mandible can be repositioned easily. Sterile gauze may be placed over the wound so that the same procedure can be carried out on the opposite side. If a bone graft is used, then the second operating team can start to remove bone from the ilium or tibia. Robinson,[3] who described a similar method, uses an iliac bone onlay. It is helpful to have available a drawing, taken from the x-ray tracings, which will show the exact size of the bone graft with measurements marked on it. In this way it is possible to remove a piece of bone larger than required, usually from the iliac crest, which can then be cut to size by the oral surgeon.

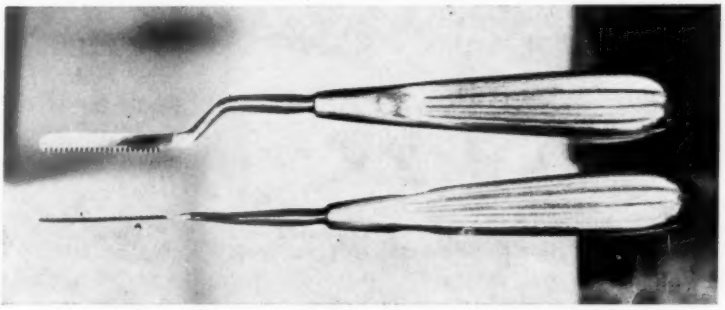

Fig. 1.—Special saws adapted for osteotomy.

While the orthopedic surgeon is removing the bone graft, the oral surgeon or a member of his team can immobilize the mandible in the acrylic splint in the new position. Care is taken, however, to keep the bone areas covered by sterile drapes during the intraoral procedure. If difficulty arises in bringing the mandible forward or in closing the anterior open-bite, it may be useful to run the index fingers back over the occlusal surfaces of the molars and create a downward and forward motion of the mandible. This will usually allow the mandible to be more easily placed in the corrected position. Intermaxillary wires and elastics may now be placed to totally immobilize the mandible. Following this, the drapes may be adjusted and gloves and gowns changed so that the areas of bone surgery can be reapproached.

The condyle and the posterior border of the ramus are now made freely movable by stripping the periosteum around it. The ramus can be placed in its proper position and held while a hole is made through both fragments with a small burr mounted in the straight handpiece. These holes should lie horizontally opposite each other or, if anything, the hole in the forward section should be placed superiorly. The bone graft can be placed and trimmed so that there is even contact of bone in all areas. The cortical layer is directed outward. A basket wire which attaches to the inner and outer portions of the horizontal wire can be placed to secure the graft. The wires are now twisted, the horizontal wire first to hold the three segments of bone in their proper position. The basket wire is twisted next. Both wires are cut ¼ inch from the bone and adapted to lie flat against it. The masseter muscle and the periosteum are repositioned and sutured with 000 plain catgut. The wound is closed in layers with 000 gut on the deeper layer and 00000 Dermalon interrupted sutures on the skin margins. Telfa is placed over the wound, and the same procedure is carried out on the opposite side. Following this, a pressure bandage is applied.

A comment regarding premedication and the type of anesthesia useful in these cases might be in order. Therefore, I have asked Dr. Robert L. Mueller (an anesthesiologist) to discuss this aspect of the operation:

"The primary goal of the anesthesiologist in surgery of this type is the simultaneous end of the anesthetic and the operation, that is, having the patient essentially awake and, if possible, free from nausea. Premedication is light, and phenergan in substituted for a barbiturate one and one-half hours prior to surgery, dosage depending on the size and age of the patient. This is repeated shortly before (that is, fifteen to thirty minutes) the end of the procedure in an attempt to reduce the possibility of nausea in the immediate postoperative period.

"Nasotracheal intubation is carried out after induction of Pentothal sodium and Anectine anesthesia, and the patient is maintained on nitrous oxide-oxygen, Anectine (1 mg. per cubic centimeter in a drip), and Pentothal sodium. These patients usually can be carried without the addition of more Pentothal sodium than was used for the intubation (400 to 500 mg.). The use of 2 per cent Xylocaine with epinephrine locally by the surgeon reduces the amount of anesthesia needed and also aids in the control of bleeding at the operative site.

"At the termination of surgery the nasotracheal tube is aspirated but left in place. It is removed by the nurse in the recovery room when the patient is fully awake and free from nausea. A suction apparatus is, of course, at hand throughout the postoperative period. Use of this general technique has enabled us to bring the patients through surgery with a maximum of safety and a minimum of complications regarding airway and respiration in general."

The postoperative care is similar to that given in cases of mandibular fractures requiring bone grafts. The jaws are left immobilized for seven to eight weeks. At the end of this period the intermaxillary wires and the acrylic splint are removed, but the fracture bars are left in place a few more days. Occlusal interferences could be adjusted after one week, but usually normal adjustments occur to make it advisable to delay this for a longer period of time. We find

that when the jaws are first mobilized the molars remain open slightly, but within one to three weeks most of this space is closed.

CASE REPORTS

CASE 1.—L. H., a 61-year-old white woman, was referred to my office on Jan. 6, 1959, for consultation regarding an anterior open-bite and associated temporomandibular joint pain.

Fig. 2.

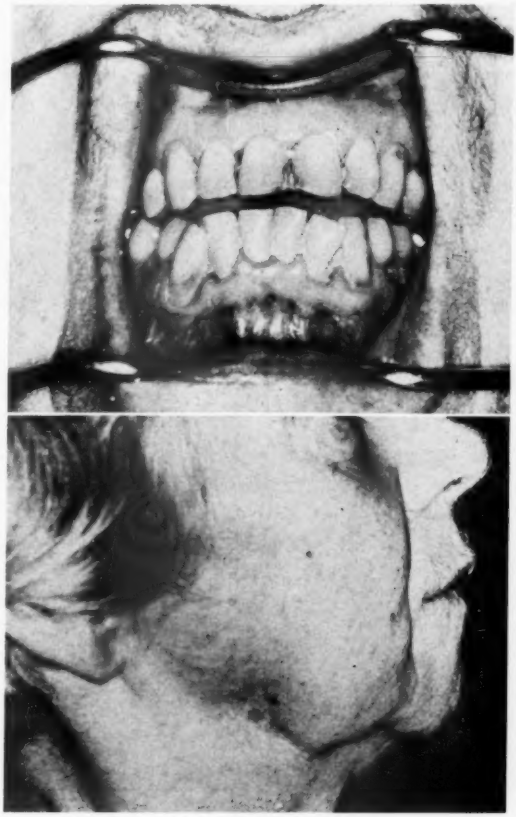

Fig. 3.

Fig. 2.—Case 1. Preoperative intraoral view of patient with open-bite.
Fig. 3.—Case 1. Preoperative profile view of patient.

Past History.—The patient's history revealed that a vertically impacted right mandibular third molar had been removed in the hospital five years previously. At the time of the surgery there was a pathologic fracture with bone displacement. The patient's oral surgeon performed an open reduction of this fracture and immobilized the fragments with an interosseous wire suture and then immobilized the mandible with intermaxillary elastic traction attached to individual Ivy loops. Following this the fracture healed, but an anterior open-bite occurred from the left first molar to the right first molar. The patient's occlusion shifted

1.0 cm. to the right, and the incisal edges of the anterior teeth were open 7 mm. During the ensuing five years the patient had a great deal of difficulty in mastication and developed definite mandibular joint pain on the right side. Weight loss, frequent headaches, and gastric and emotional disturbances developed during this period.

Over the past three years the patient had sought consultation with several general dentists and oral surgeons. Most of the treatment consisted of trying to reduce the posterior occlusion by adjusting the molar surfaces. A metal splint and also an acrylic splint were worn by the patient over the occlusal surfaces of the anterior teeth. These splints were placed to give her some surfaces on which to close her jaws, but they were very ineffective and not well tolerated by the patient.

Fig. 4.

Fig. 5.

Fig. 4.—Case 1. Preoperative oblique views of mandible showing area of fracture in right third molar area.

Fig. 5.—Case 1. Preoperative centric profile roentgenogram of patient.

Examination.—The patient was a frail-appearing woman with a moderately apparent emotional distress. She weighed 88 pounds. She stated that normally her weight had been around 110 pounds but that it had become less since her mandibular fracture. Her general health was satisfactory, with the exception of intermittent attacks of gastritis which had been controlled with medication prescribed by her physician. The laboratory findings were all within normal limits.

The oral examination disclosed an asymmetry of the face, with the mandible deviating 1.0 cm. to the right side and with the anterior incisal edges opened 7 mm. (Fig. 2). The

first teeth to occlude were the backs of the lower second premolars and the first molars. The right ramus of the mandible was angled anteriorly (Fig. 3), and there was pain to palpation over the right mandibular joint. The teeth themselves were nearly all present and in good repair.

The roentgenographic examination consisted of a centric profile view of the skull, a posteroanterior view of the skull, right and left oblique views of the rami of the mandible, mandibular joint views, and full-mouth dental views. From these views we could determine that the fracture had occurred in the right mandibular third molar area and that a wire suture remained in the area of fracture (Fig. 4). The bone in this area was thinner than normal in the vertical dimension, and a definite loss of bone in a horizontal direction caused the ramus to be angled forward. The head of the right condyle was directed posteriorly due to the forward direction of the angle of the mandible. The joint spaces were otherwise not remarkable. The centric profile view showed the severe anterior open-bite and revealed that the posterior section of the body and ramus was higher on the right side than on the left side (Figs. 5 and 6). The periapical views showed the presence of totally impacted left maxillary and mandibular third molars.

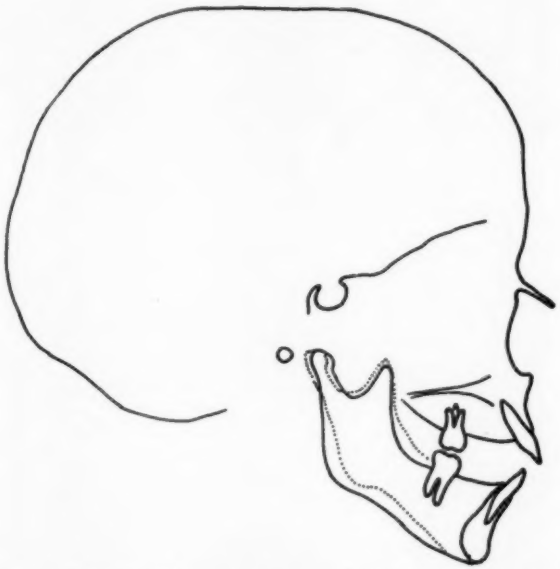

Fig. 6.—Case 1. Tracing from profile roentgenogram. Dotted line shows right mandible, while solid line shows the left.

Treatment Plan.—After the history, clinical, and roentgenographic findings had been reviewed it was decided that treatment should be directed toward correcting the anterior open-bite, the asymmetry of the mandible, and the improper position of the condyle in the glenoid fossa. A bilateral vertical sliding osteotomy of the ramus of the mandible was recommended, with an inverted V-shaped inlaid bone graft to be placed on the right side only (Figs. 7 and 8). Because of the patient's age and bone density, a tibial bone graft was thought to be most suitable.

The necessary impressions were taken, and models of the jaws were mounted in the corrected position in the articulator. The molars were placed 2 mm. apart, while the incisors were in centric occlusion and touching. An acrylic splint was prepared and fitted to the maxillary and mandibular teeth. Minor occlusal adjustments were made prior to the last impressions which were used for the fabrication of the acrylic splint.

Twenty-four hours before surgery the fracture bars were wired to the maxillary and mandibular teeth under local anesthesia.

Surgery.—On Feb. 19, 1959, the patient was admitted to St. Luke Hospital. After routine premedication, as described previously, the patient was anesthetized with intravenous Pentothal sodium and Anectine with nasoendotracheal nitrous oxide-oxygen. The acrylic splint was placed against the maxillary teeth. A 22 gauge circumferential wire was placed

Fig. 7.

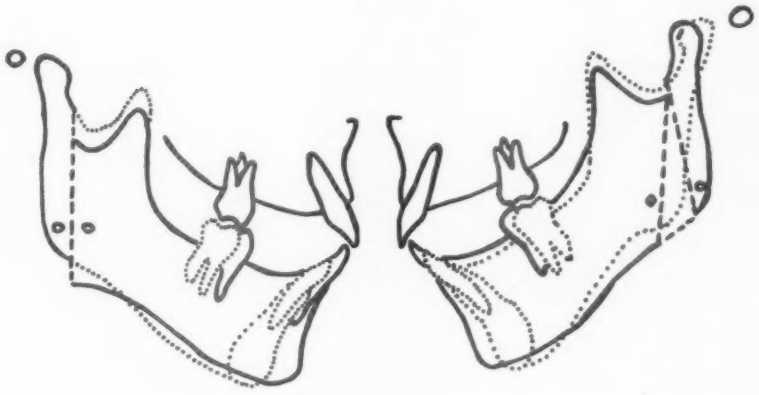

Fig. 8.

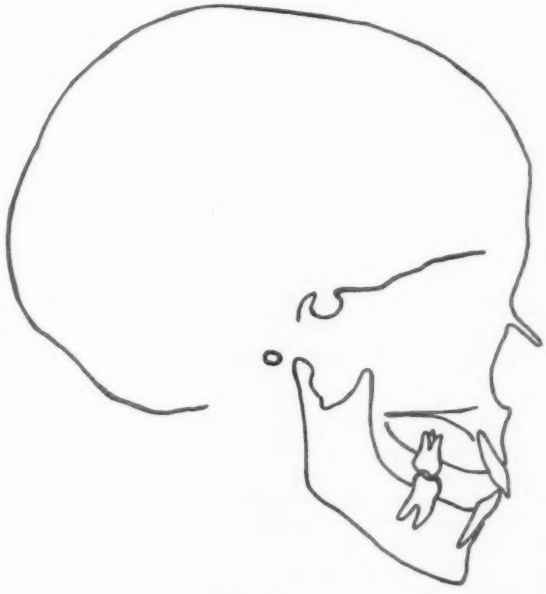

Fig. 7.—Case 1. Tracing of left and right oblique views of mandible showing proposed sliding vertical osteotomy with bone graft on right side.

Fig. 8.—Case 1. Tracing showing postoperative profile of mandible.

around the symphysis of the mandible and over the mandibular arch bar to keep it in its proper position. Two more circumferential wires were used in the mental areas.

The face and neck were rescrubbed and draped in a routine fashion. The vertical osteotomy was now performed on the left side through a 3 cm. incision in the Risdon approach. When the bone had been divided from the sigmoid notch to the angle of the mandible, a sterile pad was placed over the wound. The same procedure was performed on the right side.

Fig. 9.

Fig. 10.

Fig. 11.

Fig. 9.—Case 1. Postsurgical profile roentgenogram of mandible with jaws immobilized on acrylic splint.

Fig. 10.—Case 1. Postsurgical left and right oblique views of mandible.

Fig. 11.—Case 1. Postoperative profile roentgenogram of mandible with jaws open.

One member of the operating team working inside the mouth, brought the jaw into its proper position and immobilized it with steel wires between the fracture arch bars. During this time the orthopedic surgeon removed a triangular piece of bone from the medial aspect of the left tibia. The drapes were now adjusted and the bone graft was trimmed and placed in position on the right side, allowing the condylar section to take a normal direction. Two 24 gauge steel wires were used to immobilize the graft and the fragments in their properly adapted positions (Figs. 9 and 10). The wound was closed in layers and covered with Telfa. The left osteotomy site was now inspected and the fragments were adapted properly and fastened with one 24 gauge interosseous steel suture. The wound was handled in a manner similar to that employed on the right side. Pressure pads were placed over all areas of surgery.

The postoperative course was uneventful. Skin sutures were removed on the seventh day, and immobilization of the jaws was discontinued during the eighth week. The jaws began to function correctly almost immediately and within a week or two the patient had a normal amount of vertical opening (Fig. 11) and was able to make all lateral and protrusive movements.

Fig. 12.—Case 1. Postoperative intraoral view of patient's corrected occlusion.

The posterior occlusions remained slightly open for three to four months, and then a slight equilibration of anterior occlusion was done to allow all teeth to contact in centric occlusion.

The patient's ability to eat became normal, her normal weight returned, and her mandibular joint pains disappeared completely. Her gastritis has not returned to date.

Her right condyle and ramus are now identical in position to those on the left side. The facial asymmetry was improved, and the open-bite was corrected (Fig. 12).

CASE 2.—K. B., a 21-year-old senior student in the nursing program at Huntington Memorial Hospital, was referred to my office on Feb. 5, 1959, for appraisal and treatment of her underdeveloped chin.

Examination disclosed slight underdevelopment of the symphysis of the mandible and a very noticeable anterior open-bite extending from the second premolar regions bilaterally. The mandible was retruded approximately 7.5 mm., and the open-bite measured 1.0 cm. (Fig. 13). Maxillary and mandibular protrusion of teeth was severe, causing a perioral bulging, and the chin was unbecomingly retruded (Fig 14). There was no visible angle of the mandible.

The past history and general physical examination were not remarkable.

The roentgenographic survey included a centric profile view of the skull, a postero-anterior view of the skull, right and left oblique views of the rami of the mandible, and full-mouth periapical views.

These films showed a retrusion of the mandible with marked protrusion of the anterior teeth causing the lips to protrude severely. The mandibular occlusion had a steep downward plane inclination in a posteroanterior direction. There was also a rather marked curve of Spee. Study models were now made from accurate impressions of the dental arches, and they confirmed the anterior open-bite and retrognathia.

Fig. 13.

Fig. 14.

Fig. 13.—Case 2. Preoperative view of patient's occlusion showing an anterior open-bite.
Fig. 14.—Case 2. Preoperative profile view showing retrusion of mandible.

At this point, consultation was held with an orthodontist, Dr. George Boone. From tracings made from the centric profile roentgenogram, the case could be studied with respect to treatment from purely an orthodontic viewpoint. It was decided that the four first premolars could be removed in order to retrude the maxillary teeth, but this would not produce the desired chin and it would not entirely close the anterior open-bite. It was felt that the contemplated orthodontic therapy would not give the optimum results. At this point the case was evaluated further from the standpoint of oral surgical correction. It was decided that a bilateral vertical sliding osteotomy (Fig. 15) of the ramus of the mandible, with the placing of bone grafts (Fig. 16) would correct the anterior open-bite, elongate the mandible, and improve the occlusal plane (Fig. 17). It was believed that when the surgery had been

completed orthodontic treatment of the kind mentioned above would produce a proper intercuspal relationship of the teeth and reduce the protrusion of maxillary and mandibular teeth and lips.

Selective equilibration in the first premolar areas was done, after a study of the models, so that the most contact of teeth could be accomplished. An acrylic splint was constructed after the models had been set up in the desired position with the molars open 2.0 mm. and the incisors contacting. The splint was placed first against the maxillary teeth and then against the mandibular teeth to check for fit.

Twenty-four hours before surgery the maxillary and mandibular fracture bars were placed in position under local anesthesia.

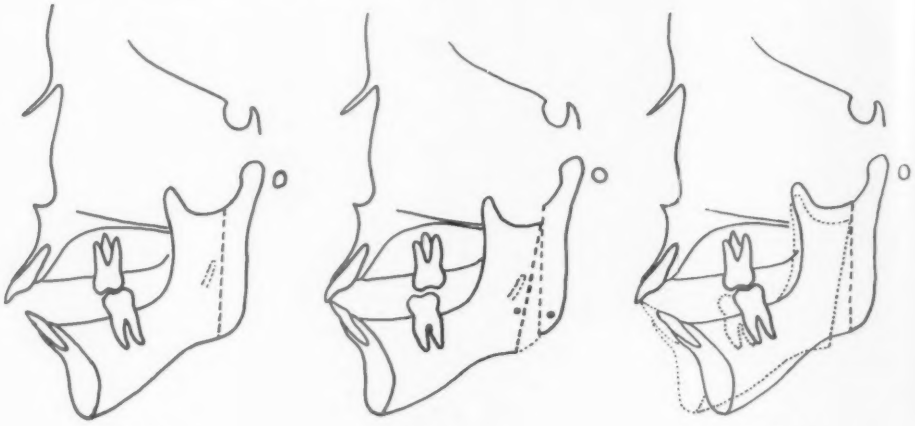

Fig. 15. Fig. 16. Fig. 17.

Fig. 15.—Case 2. Tracing showing direction of osteotomy.
Fig. 16.—Case 2. Tracing showing mandible moved forward and area for bone graft.
Fig. 17.—Case 2. Tracing showing amount of change occurring after jaw is moved forward.

Surgery.—On April 12, 1959, the patient was admitted to the hospital. The following morning she was given routine premedication and later anesthetized with intravenous Pentothal sodium and Anectine with nasoendotracheal nitrous oxide-oxygen. Three circumferential wires were placed around the mandible to hold the mandibular fracture bar in its proper position, and the acrylic splint was placed against the maxillary teeth. The operation was identical to that performed in Case 1, except that the triangular bone grafts were used on both sides (Fig. 18). The bone was taken from the crest of the left ilium by the orthopedic surgeon.

The patient's postoperative course was uneventful. She returned to her nursing duties on the tenth postoperative day. At the end of the seventh postoperative week, the intermaxillary traction was eliminated. The patient was immediately able to open her jaws and to achieve lateral and protrusive movements. One week later the fracture bars and circumferential wires were removed under local anesthesia.

Ten days following mobilization of the mandible, the patient was able to open her jaw approximately 1¼ inches and had no sensations of pain, pressure, or pull. The patient has continued to open her jaws more fully.

The anterior occlusion closed, and after four weeks the molars had drifted together so that there was contact of all the teeth from the right second molar to and including the left first molar. The patient's occlusion was excellent (Fig. 19), and her profile and full face had a much more pleasing appearance than before (Figs. 20 and 21). At the present time the patient is under orthodontic care designed to complete the treatment and produce the best possible results.

COMMENT ON THE TECHNIQUE

A technique for improving the occlusal plane, closing open-bite, and lengthening the mandible has been presented. Among the advantages of this relatively

Fig. 18.

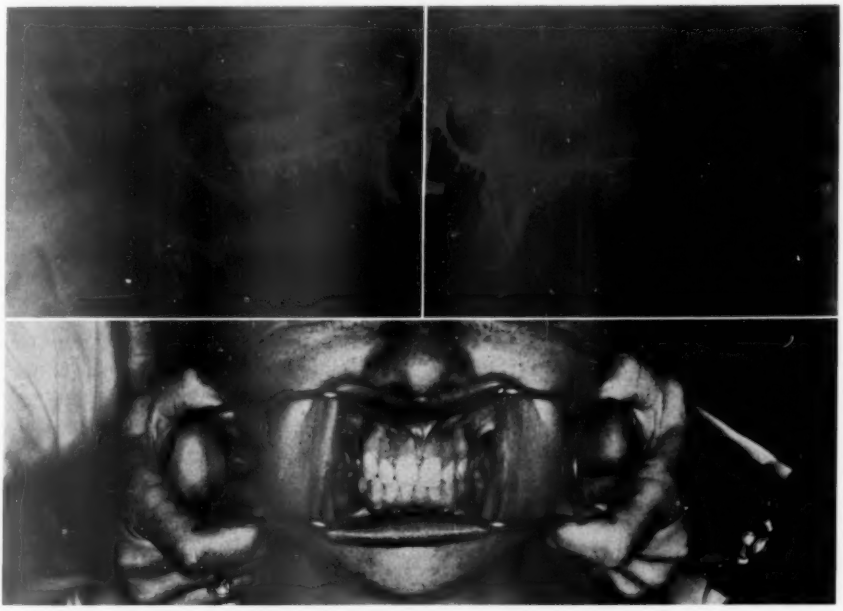

Fig. 19.

Fig. 18.—Case 2. Postsurgical roentgenograms of rami of mandible showing bone graft.
Fig. 19.—Case 2. Postoperative view of patient's occlusion.

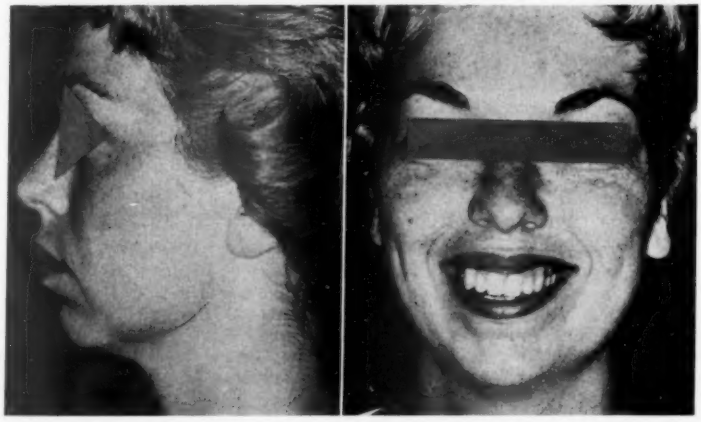

Fig. 20. Fig. 21.

Fig. 20.—Case 2. Lateral view of patient three months after surgery.
Fig. 21.—Case 2. Final postoperative view of patient.

simple one-stage operation are the following: (1) it does not disturb the nerves or vessels of the area, (2) it does not require removal of teeth, (3) it corrects occlusion, and (4) it produces a more esthetic position for the chin and the angles of the mandible. The direction of the osteotomy is such that the greatest amount of closure of the anterior occlusion, with the least amount of separation of bone fragments and the greatest length of bone contact for healing, is available. If the mandible is to be lengthened horizontally, then the muscle pull on the fragments will have no chance for separation but will tend to pull the fragments more firmly together. The area of osteotomy is an area of rapid healing and in no way disturbs the mandibular joint. Perhaps some speculation regarding the effect of stretching the temporal muscle is in order, but no harmful effect, such as a lack of jaw opening, has been noted. If this should become a problem, however, an intraoral bilateral postsurgical coronoidectomy would correct it.

The frequency of retrognathia associated with a steep occlusal plane or an anterior open-bite makes this procedure most advantageous. To relieve the "Andy Gump" chin and mandible, a genioplasty might be performed later.

The construction of the occlusal splint and the use of at least one anterior mandibular circumferential wire are a most desirable, if not mandatory, part of the procedure. Proper roentgenographic studies are imperative. Proper premedication, anesthesia, and pre- and postoperative treatment are necessary to reduce the possibility of untoward sequelae. Certainly, a careful explanation of the over-all treatment plan to the patient is to be recommended.

SUMMARY

The surgical procedure of a vertical sliding osteotomy of the ramus of the mandible for correction of retrognathia and anterior open-bite has been presented. To correct retrognathia, an inlaid bone graft from the iliac crest or from the tibia is used.

A case of mandibular asymmetry and anterior open-bite in a 61-year-old woman and another case of retrognathia and anterior open-bite in a 21-year-old woman, both corrected by this operation, have been presented.

Since no conclusions can be drawn from two cases treated in this manner, there will be frequent examinations of these patients and a follow-up of this preliminary report.

In order better to record and correlate the total progress of these cases, I have had color motion pictures of these patients taken before, during, and after surgery. This will permit the utmost accuracy in a future final report.

REFERENCES

1. Thoma, Kurt H.: Personal Communication, 1959.
2. Thoma, Kurt H.: Oral Surgery, ed. 3, St. Louis, 1958, The C. V. Mosby Company, p. 1407.
3. Robinson, Marsh: Micrognathism Corrected by a Vertical Osteotomy of Ascending Ramus and Iliac Bone Graft: A New Technique, ORAL SURG. ORAL MED. & ORAL PATH. 10: 1125-1130, 1957.
4. Sicher, Harry: Personal Communication, 1959.

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DIAGNOSIS AND TREATMENT OF A STYLOID FRACTURE

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FRACTURES of the styloid process have not often been reported in the dental literature. The case to be presented here will serve to demonstrate the diagnosis and treatment of a styloid fracture.

ANATOMY OF THE AREA

The styloid process leaves the temporal bone immediately behind the mandibular fossa, which is located in front of and below the auditory meatus. The styloid process serves as the attachment of the styloglossus, stylohyoideus, and stylopharyngeus muscles and the stylomandibular ligament.

Fig. 1.—Lateral plate of left side of mandible revealing a fracture of the styloid process.

CASE REPORT

The patient, a 33-year-old white married woman, complained of severe pain in the left side of the throat and lower left molar area. She stated that the pain increased when she swallowed or moved her jaw from side to side.

She gave no history of a recent fall or of trauma to the left side of the face. One year previously a lower left third molar had been removed, following which the patient experienced severe facial swelling and inability to open her mouth.

Extraoral examination revealed nothing of clinical significance. Intraoral examination of the left third molar region revealed a normal mucosal covering of the area. The soft palate of both sides was palpated with digital pressure. As the examining finger was passed laterally and posteriorly into the soft palate and tonsillar fossa on the left side, the patient began to experience severe pain in the throat and in the left side of the mandible. This pain was considerably relieved when the pressure was released.

Roentgenographic Examination.—An oblique lateral plate (Fig. 1) of the left side of the mandible revealed a fracture of the styloid process.

Treatment.—The patient was placed on Darvon, 65 mg. every six hours as needed. The posterior sections of the maxilla and mandible were wired and rubber band elastics were applied. The patient was instructed to remove the elastics only at meal time. She was further instructed to eat only those foods which required a minimal amount of chewing.

With the jaws wired together, the pain gradually diminished. At the end of ten days the patient discontinued the Darvon. After five weeks the wiring was removed. The patient refused postoperative roentgenograms and, in view of the normal function and the lack of pain, she was discharged.

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EXODONTIA

RETENTION OF A LOWER FIRST PERMANENT MOLAR

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RETAINED teeth are found not infrequently in dental practice. Although most textbooks do not give statistics as to their occurrence, mention is often made of the frequency with which various teeth are found to be retained.

Thoma[7, 8] and Stones[6] refer to statistics which Blum reported in 1923. According to those figures, 86 per cent of all malposed mandibular teeth are third molars. Two retained first mandibular molars were found in a series of 267 cases, but in the same series there was not a single case of first- or second-molar retention in the upper jaw.

According to Archer,[2] the teeth most frequently retained are (in decreasing order) the lower and upper third molars and the upper and lower cuspids. Archer states that impaction of the lower first molar is very rare.

Euler,[3] in 1939, mentioned a compilation made by Luniatschek in 1906 and reproduced a tabulation of retained teeth that Prinzing (1935) observed during a three-year period in Marburg. The latter work does not mention a single case in which a lower first molar was retained, whereas retention of one upper first molar and two lower second molars is reported.

This brief review of the literature indicates that first-molar retention is rather rare. Therefore, it is believed that the following case will be of interest.

CASE REPORT

A 19-year-old male patient, employed as a tailor, was admitted to our outpatient department on Sept. 23, 1959. Four days before coming to the clinic he had a chill. High fever followed, and the patient complained of gradually increasing pain in the left lower quadrant of the face. The next day this region became swollen. Because of the increasing fever and marked swelling, the patient sought advice at our clinic.

Findings on Admission.—The patient's skin was moist and hot. His pulse rate was accelerated. There was a medium-sized swelling of the left lower face. On intraoral inspection, no tooth was seen behind the left lower premolars, with the exception of the third molar

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which was markedly tilted. There was a painful gingival swelling back of the second premolar. This swelling was livid red in color, and application of pressure elicited a discharge of pus through a small fistula.

Roentgenographic examination disclosed a tooth deeply embedded in the bone (Fig. 1). Because of its position in relation to the other teeth, this was assumed to be the first permanent molar. It is noteworthy that the occlusal surface of this deeply embedded tooth was markedly destroyed. On the basis of roentgenographic and clinical findings, a diagnosis of purulent osteitis associated with a retained first permanent molar was made.

Treatment.—On admission to the clinic, the patient was given 200,000 units of penicillin three times daily. Under conduction anesthesia, the embedded tooth was removed via an intraoral approach. Because the tooth was very deeply embedded, it was necessary to remove the second premolar also. In spite of the rather extensive bone operation in an infected area, recovery was uneventful.

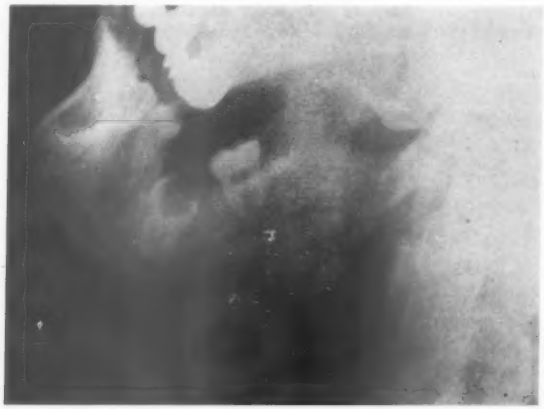

Fig. 1.

DISCUSSION

Retention of a first molar is rare. In his book entitled *Oral Pathology,* Thoma[7] reproduces a roentgenogram by Blum who himself observed two cases of first-molar retention. Thoma[8] mentions one case in *Oral Surgery.* Izard[4] refers to a single observation by Cadenat of a retained lower first molar, and Adler[1] reports a case in which an upper first molar was retained.

Our knowledge concerning the etiology of tooth retention is rather limited. Textbooks usually present an intricate system of etiological factors in which all possibilities are included. Lack of space and early loss of the deciduous teeth (especially from the "support zone") are generally accepted causes of retention. Similarly, it is assumed that lack of space might cause retention of the mandibular third molar in the shortened body of the lower jaw. Sometimes reference is made to modern man's diet, which does not require vigorous mastication and thus does not act as a stimulus to bone growth. There is no positive proof of this view, although some findings (for example, the nonoccurrence of tooth retention among the Bedouins of the Negev Desert and among Greenland Eskimos) seem to give it support.

Archer[2] distinguishes between local and systemic causes and mentions that retention in rare morbid conditions is a separate entity. This classification was not of much help to us either.

It is impossible to decide what was the ultimate cause of tooth retention in our patient. Prenatal influences can be excluded with certainty. As postnatal causes, eruptive diseases are often mentioned. In our patient we were unable to rule out such a cause, but we were also unable to prove any connection between a disease of this kind and the abnormal condition of the first molar. The roentgenogram failed to show any pathosis or any sign of a precedent pathologic process in the vicinity of the retained tooth. The rare morbid conditions (for example, oxycephaly, progeria, cleidocranial dysostosis) are to be excluded also, since no symptoms were detected. No data were found as to familial occurrence of tooth retention. Lack of space could hardly have caused the retention, since there was an empty space for at least one extraordinarily large molar between the second premolar and the forward-tilted third molar. The second molar, which erupted normally, had been extracted some time earlier.

We must frankly admit that no obvious cause for retention of the first molar was found in our patient. In this case, one is wont to speak of idiopathic tooth retention. Naturally, this is not an etiological diagnosis. On the contrary, it is a frank admission of failure to unravel the problem of etiology. This failure is not unusual. In his recent handbook on dentistry, for example, Meyer[5] states that the etiology of the rare first- and second-molar retentions is unexplainable.

In the case described here the retained tooth caused one of the more unusual complications, namely, a periodontal infection resulting in mandibular osteitis. We feel justified in assuming that the infection occurred primarily through the empty socket after the second molar was extracted. The deep destruction of the occlusal surface of the retained tooth was an extraordinary finding (Fig. 1). This cannot be distinguished from a carious tooth destruction. Destruction of the occlusal surface is indicative of a communication between the crypt of the retained tooth and the oral cavity proper. Hard-tissue destruction was so extensive that the pulp of the retained tooth was found to be gangrenous.

No signs of periapical involvement were seen around the retained tooth in the roentgenogram (Fig. 1). This negative finding does not entirely exclude the possibility that the osteitis was caused by a periapical infection of the retained tooth. According to the patient's history, there was no previous inflammatory condition. With the first involvement of periapical tissues, roentgenographic changes are not seen for several days. It seems more probable, however, that the osteitis started from the pericoronal infection, much as it does from half-impacted mandibular third molars.

REFERENCES

1. Adler, P.: Ein retinierter Sechsjahrmolar, Deutsche zahnärztl. Wchnschr. 41: 379, 1938.
2. Archer, W. H.: Manual of Oral Surgery, Philadelphia, 1952, W. B. Saunders Company, pp. 82-83, 112.
3. Euler, H.: Die Anomalien, Fehlbildungen und Verstümmelungen der menschlichen Zähne, München, 1939, J. F. Lehmanns Verlag, p. 108.

4. Izard, G.: Orthodontie, Paris, 1950, Masson & Cie, p. 538.
5. Meyer, W.: Anomalien der Stellung einzelner Zähne. In Häupl, K., Meyer, W., and Schuchardt, K.: Die Zahn-, Mund- und Kieferheilkunde, Berlin, 1958, Urban & Schwarzenberg, Bd. I, p. 704.
6. Stones, H. H.: Oral and Dental Diseases, London, 1957, E. & S. Livingstone, pp. 94, 112-130.
7. Thoma, K. H.: Oral Pathology, St. Louis, 1950, The C. V. Mosby Company, p. 212.
8. Thoma, K. H.: Oral Surgery, St. Louis, 1948, The C. V. Mosby Company, pp. 315-317.

ORAL MEDICINE

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HERPES ZOSTER—A PRIMARY MANIFESTATION OF CHRONIC LYMPHATIC LEUKEMIA

Report of a Case

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HERPES ZOSTER is the term assigned to the clinical manifestations resulting from irritation of a posterior root ganglion of a spinal nerve or an extramedullary ganglion of a cranial nerve. The disease is characterized by burning, pruritus, acute neuralgic pain, and vesicular eruption in the area of anatomic distribution of the peripheral sensory nerves arising in the affected root ganglion.[1]

ETIOLOGY AND INCIDENCE

Herpes zoster has been classified as primary and secondary in origin. It is thought that primary herpes zoster occurs directly as a result of infection of a posterior root ganglion.[2] Rosenow and Oftedal[3] expressed their belief that herpes zoster resulted from hematogenous streptococcal infection of a posterior root ganglion. McEwen[4] and others feel that the virus of chickenpox, varicella, or a virus closely related to varicella is the causative agent in herpes zoster. Perusal of the literature has not revealed definitive substantiation for either theory. The viral theory, however, is strengthened by reports of the simultaneous occurrence of chickenpox and herpes zoster in the same household.[5] Cecil and Loeb,[2] in their *Textbook of Medicine,* state: "Varicella has been produced experimentally in children by intradermal inoculation with zoster vesicular fluid."

Secondary herpes zoster has been reported to occur as a complication of debilitating diseases, such as pneumonia, tuberculosis, uremia, and nutritional deficiencies; as the result of trauma, such as fractures of the spine and cranium and dislocations of the spine; from poisoning by carbon monoxide, arsenic, and bismuth; from irritation and/or impingement by neoplasms, subarachnoid hemorrhage, and cellular infiltrations (leukemias); and as a

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complication of syphilis and meningitides.[1, 2, 6, 7] Decayed[8] and supernumerary[1] teeth have been said to have a causal relationship, since in some instances pain and vesiculation have disappeared upon their removal.

Incidence.—In spite of the numerous etiological possibilities, herpes zoster is a relatively rare disease. It occurs more frequently in the male than in the female,[9] and although it may occur at any age it is most common in the elderly.[7] The disease apparently has a seasonal predisposition, since it occurs most often during cold, damp weather of early spring and late autumn.[6] The seasonal incidence and the age of the patients indicate a prevalence of the disease in persons who are overworked, fatigued, malnourished, or ill.[1]

Site of Involvement.—Berggreen and Schüler[10] studied the sites of involvement in 2,014 cases of herpes zoster. The trigeminal nerve was involved in 16 per cent of the cases, the cervical nerves in 25 per cent, the dorsal spinal nerves in 48 per cent, the lumbar nerves in 9 per cent, and the sacral nerves in 2 per cent. Of the cranial nerves, the ophthalmic division of the trigeminal nerve was involved most frequently, followed by the maxillary and mandibular divisions in the order named.[1] Affliction of the ophthalmic division of the trigeminal nerve may affect the cornea, resulting in corneal opacities as well as dermal lesions of the forehead.[1] Involvement of the maxillary or mandibular divisions of the trigeminal nerve may result in lesions on the oral mucosa and skin, or on both simultaneously.[1] The most frequent intraoral sites are the anterior portion of the tongue, the soft palate, and the cheek. Multiple involvement of the fifth, seventh, eighth, and ninth cranial nerves may occur.[11] Involvement of the geniculate ganglion of the seventh cranial nerve results in production of the so-called Ramsey-Hunt syndrome, which consists of herpes zoster, otic pain, vertigo, nausea, vomiting, gustatory disturbance, diminished hearing, and occasionally a bloody discharge from the external auditory meatus.[1, 6] The otic involvement is mediated through the ganglionic relationship to the eighth cranial nerve. Complete paralysis of the seventh and eighth cranial nerves may also occur.

Symptoms and Gross Pathology.—Premonitory symptoms, both constitutional and local, precede the eruption of the vesiculation of herpes zoster. Constitutional disorders, such as malaise, fever, headache, and dizziness, can occur prior to or concomitant with local symptoms of hyperthesia, itching, and a burning sensation.[1, 6] Involvement of one of the cranial nerves may also be indicated by facial muscle spasm.[6] Regional lymphadenopathy may be present as a premonitory finding, or it may become apparent at the time of vesiculation.[2]

Following a short, variable period of premonitory symptoms, vesiculation occurs along the distribution of the peripheral terminations of the affected nerve or nerves.[2] The vesicles develop upon a broad and slightly elevated erythematous base and are accompanied by neuralgic or acute, deep, burning pain.[1] The vesicles formed are filled with a clear serous fluid. Vesiculation is primarily unilateral, although cases of bilateral involvement have been reported.[12] The vesicles persist for a variable time, depending upon extrinsic

factors of environment, clothing, sleeping habits, and toilet care as well as intrinsic healing factors. Dermal vesicles may persist as long as ten days, while those that occur in the oral cavity are of short duration (hours).[1] The vesicles, when present in the oral cavity, are similar and clinically indistinguishable from those found in herpes simplex. Rupture of the oral vesicle leaves a lesion which is viewed as an eroded area within an erythematous ring. Ragged epithelial remnants of the ruptured vesicle walls are found attached at the periphery of the eroded mucosa. Within several days the erosion takes on a yellow-gray, cheesy coating which persists during repair of the underlying mucosa.[1] The pain that accompanies the oral lesions is at first of minor consequence, but when the vesicles rupture the pain becomes acute. The onset of severe pain is thought to be due to exposure and irritation of the eroded mucosa and nerve endings by saliva, air, food, bacteria, and subsequent secondary infection. The oral lesions of herpes zoster are of shorter duration than the dermal lesions.[1] Residual scarring is infrequent.

Fever occurs concomitant with the onset of dermal vesiculation, and with its subsidence, usually within three to five days, drying or rupture of the vesicles takes place.[2] Rupture of the dermal vesicle produces a hard, dark, crustaceous scab due to drying of the serous and occasionally bloody discharge of the lesion. Secondary infection usually results at this time. Similar to other lesions affecting the skin, when the scab is lost the repaired lesion presents a white, ischemic, and fibrous appearance. Permanent scarring of the skin may occur following severe infection, but the majority of cases proceed to a normal tissue appearance.[2] The pain accompanying dermal lesions is intense and deep-boring in nature, and it may persist after resolution of the lesions.[2]

Histopathology.—In herpes zoster, "as in herpes simplex, we find epithelial degenerations with the formation of vesicles beneath which there is a marked inflammatory reaction. The eosinophilic bodies which occur in herpes simplex are also found in herpes zoster. The vesicles are formed in the epidermis by swelling and rupture of the epithelial cells. The intercellular inclusion, the so-called 'zoster' or 'Lipschütz body' may be seen in the sections. These are swollen cells, also named balloon cells, probably produced by nuclear degeneration caused by the virus."[6] "The lesions in the ganglia are similar to those in the anterior horn in poliomyelitis, i.e., congestion, hemorrhage, perivascular collections of lymphocytes, and degeneration of ganglion cells with neuronophagia. There is Wallerian degeneration of the nerve fibers in the posterior roots, in the peripheral nerves, and in the posterior columns."[13]

CASE REPORT

At the request of his physician, Mr. J. B., a 60-year-old white man, was seen on Oct. 1, 1954, in the Oral Surgery Department of the Millard Fillmore Hospital in Buffalo, New York.* His chief complaint was acute and constant oral and facial pain of approximately eight days' duration, accompanied by inability to eat, drink, sleep, and perform daily oral and facial hygienic measures comfortably.

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Present Illness.—Seven days previously, on Sept. 24, 1954, the patient was told upon his return home after obtaining a haircut, that the left side of his face presented multiple splotchy areas of redness. The patient was unaware of the condition but had noticed a feeling of warmth and itchiness on the left side of his face, which he attributed to his tonsorial care. Mirror examination confirmed the family's observation, but the patient did not become alarmed. He was able to ingest his dinner without difficulty, but as the evening progressed he became increasingly uncomfortable. His family noticed that the facial splotches were undergoing a change. The patient was unable to describe them. Following a restless and uncomfortable night, blisters were observed the next morning on the left side of the patient's face and on the tip of his tongue. His discomfort had increased greatly, and he was unable to shave or to take solid nourishment. Attempts to alleviate his discomfort with proprietary analgesics were only partially successful.

Fig. 1.—Left lateral view of dermal lesions (eighth day). Note lesions extending into buccal and auriculotemporal nerve distribution.

Later in the day (Sept. 25, 1954) the patient consulted a physician, who prescribed a salve for the facial lesions. Consideration was given to relief of pain and to dietary care. The patient took the prescription to a pharmacist friend, who quickly informed him that the salve would be of little value and promptly prescribed and dispensed his own remedy. The patient followed the pharmacist's advice and took the medication without improvement or relief. In fact, the pain and discomfort increased and the diet was painfully limited to liquids. By the fifth day, the dermal vesiculations had ruptured and become "scabby." On the evening of the sixth day, the patient was seen by a general surgeon who found, in addition to the dermal and oral lesions, a tumefaction in the left maxillary vestibule in the premolar-molar region. Upon questioning by his physician, the patient revealed that his denture fit poorly and that it had been constructed by a dental technician friend without the benefit of attention by a licensed doctor of dentistry. Because of the tumefaction and oral lesions, the physician referred the patient to the Oral Surgery Department of the Millard Fillmore Hospital for consultation and diagnosis.

Past history was noncontributory. The patient stated that he had been in good health until the present illness.

Findings.—The patient, when seen on Oct. 1, 1954, was in acute distress, moderately cachectic, and dehydrated. Malaise was prominent. Examination revealed multiple darkly

encrusted lesions on a broad erythematous base localized to the anatomic distribution of the mandibular division of the left trigeminal nerve. Facial lesions were present in the left preauricular, paramandibular, and lateral inferior aspects of the cheek, in the inferior lip and mental region to the midsagittal plane, and in the parietal region (Fig. 1). The lesions were secondarily infected and varied in diameter from ½ inch to 1 inch. Orally, there were

Fig. 2.—Magnified view of labial and tongue lesions (eighth day).

Fig. 3.—Anterior view of dermal and oral lesions at eight days. Note that lesions stop at midline.

multiple aphthae with a cream-colored plaque center encircled in some instances with epithelial tags and in all instances with an erythematous ring. The oral lesions were limited to the left side of the tip and anterior third of the tongue and to the cheek mucosa in its posterior aspect. The tongue was heavily coated in the middle and posterior thirds (Figs. 2 and 3). The maxilla was edentulous. The patient stated that he had a maxillary denture but had been unable to use it since the onset of his illness. An extensive granuloma fissuratum was present

in the left maxillary vestibule in the cuspid-to-molar region. The patient stated that the "growth" had been present for a minimum of six months. Five teeth, all periodontally involved, comprised the mandibular dentition. The patient was without a mandibular prosthesis. Salivation was minimal, but thick and stringy. The oral tissues showed evidence of dehydration.

The left submaxillary, preauricular, submental, superficial, and superior deep cervical and supraclavicular lymph nodes were enlarged, nonmatted, and tender (Figs. 1 and 3). The lymph nodes on the right side were not remarkable. The patient was questioned with regard to eruptions elsewhere on his body and the possibility of a previous eruption. He denied the latter but stated that there was a rash on his back at the time. The patient associated the rash with perspiration resulting from working in his yard during several days of hot weather approximately two days prior to the onset of his present illness. He stated that he perspired freely and was highly susceptible to heat-induced rashes. There was no evidence of vesiculation, but there was a red rash on his back, extending to the medial supraclavicular region in an open V-neck manner. The axillary lymph nodes were not remarkable.

Fig. 4.—Anterior view of dermal lesions following x-ray treatment. Twenty-three days after onset of illness.

Roentgenograms of the skull were ordered to rule out neoplastic impingement upon the trigeminal nerve. The x-ray findings were reported to be within normal limits.

Treatment.—The oral cavity was gently irrigated with warm water, and aqueous Merthiolate was applied to the oral lesions. The oral cavity was then irrigated with a warm solution of Maalox. The Maalox rinse brought comfort to the patient, and he was advised to use it at home every two hours. Therapeutic Becotin with C three times daily was prescribed as a dietary adjunct. A dietary regime was established to ensure adequate nourishment. Analgesics were not prescribed, since this had been done previously by the referring physician.

A diagnosis of herpes zoster involving the mandibular branch of the left trigeminal nerve was made. The referring physician was contacted by telephone and given the diagnosis along with the recommendation that the patient be directed to a dermatologist for x-ray therapy. He agreed and the patient was referred to a dermatologist although he was to be followed at the oral surgery department as well.

The diagnosis of herpes zoster was confirmed by the dermatologist on Oct. 2, 1954. His report of treatment follows:

" . . . He was given three x-ray treatments consisting of 100 roentgens filtered through 1 mm. of aluminum with the factors of 100 kv. and 5 Ma. to the preauricular ganglion on the left cheek. This dosage was administered on October 2, 5, and 8. At the time of his last examination on October 15, the eruptions were subsiding satisfactorily, but Mr. J. B. still continued to have some residual pain in the area. He was returned to the care of his referring physician and not seen after that time."[14]

During the period of x-ray treatment, the patient did not keep his appointments at the oral surgery clinic. Telephone conversations elicited the information that his oral and general condition was improving. The patient was seen on Ocobter 15 after his visit to the dermatologist. The lesions were receding (twenty-three days since the onset of herpes zoster), and he was "fairly comfortable." Gross left submaxillary and preauricular swelling was present but nonpainful (Fig. 4). A checkup appointment was made for the next week, but the patient failed to keep this and other appointments during the following three months. On Jan. 15, 1955, the patient's wife stated, during a telephone conversation, that Mr. J. B. was not eating well, was sleeping poorly, and had constant gastrointestinal discomfort. An appointment was made and kept by the patient on Jan. 16, 1955. Examination revealed a grossly apparent weight loss. The lesions of the herpes zoster were well healed, with little scarring. The granuloma fissuratum was reduced in size and in inflammatory response. The facial contour was within normal limits; however, examination of the lymph nodes of the head and neck revealed marked bilateral enlargement. The nodes were not matted or tender. The patient was asked to remove his shirt, and the axillary nodes were grossly evident upon palpation. There was no evidence of a dermal rash upon the back or chest. The inguinal nodes were enlarged to the extent that they were palpable through the patient's trousers as well as in the conventional manner. The patient was immediately taken to the hematology laboratory for a complete blood count. On the basis of the clinical findings and history, a diagnosis of leukemia was made. To be ruled out were Hodgkin's disease, lymphosarcoma, and infectious mononucleosis. The patient's referring physician was contacted by telephone and advised of the recent findings and the tentative diagnosis. The results of the complete blood count done on January 18 were as follows:

Red blood count	4,770,000	Hemoglobin 14.4 Gm. 99 per cent; 14.5 Gm. 100 per cent
White blood count	370,000	Platelets may be slightly decreased
Filaments	2%	Occasional young lymphocytes
Bands	-	Occasional small lymphocytes
Intermediate lymphocytes	85-90%	Slight anisocytosis
Prolymphocytes	5-8%	Occasional smudge cells

A diagnosis of chronic lymphatic leukemia was made by the hospital hematologist. A copy of the report was sent to the referring physician, and the case was discussed with him by telephone. The patient was then contacted and advised to see his physician for treatment. The patient was seen by the referring physician on Jan. 17, 1955, at which time he was advised that he was seriously ill and required extensive therapy. The patient, however, was not impressed and refused to undergo treatment. In view of this attitude, his physician told him that he had leukemia. Surprisingly, the patient still refused treatment, saying that he had lived a good lengthy life and that he did not expect to live forever. At his family's insistence, he finally agreed to undergo treatment at the Roswell Park Memorial Institute, a cancer research and treatment center in Buffalo, New York. Treatment was instituted on Feb. 1, 1955. A treatment report dated June 22, 1956, follows:

> . . . His first treatment consisted of steroid treatment and divided doses of triethylene malamine for a total dosage of 25 mg. This therapy reduced the W.B.C. from 590,000 to 121,000. Since that time, he has had infrequent ingestion of triethylene malamine and prednisone . . . which has reduced the size of the liver and spleen. From April 5, 1955, to April 19, 1955, he received irradiation therapy

to the spleen. He was last examined on June 7, 1956, at which time the blood findings were hemoglobin 11.2 Gm., W.B.C. 125,000, 96 per cent lymphocytes, platelets 88,000. He has been ingesting prednisone, 5 mg. twice daily, since about May 1.[15]

The patient was reported active and well in the fall of 1957.

DISCUSSION

In the case just described herpes zoster was a primary symptom of lymphatic leukemia. As far as can be ascertained, this case is the first of its type reported in the dental literature and the thirty-eighth case of concurrent herpes zoster and leukemia reported in the general medical literature during the past sixty-seven years. Although herpes zoster is not common, Berggreen and Schüler's[10] evaluation of 2,014 cases reveals a 40 per cent incidence in the head and neck region. The etiological factors, both local and systemic, previously listed in conjunction with the high relative percentage of herpes zoster in the head and neck area should give justification for the dentist to be aware of the ramifications as well as the primary aspects of the disease. Although the primary or "viral" form is most often seen, serious consideration and evaluation should be directed to the possibility that the herpes zoster is secondary to systemic disorders or localized ganglionic and nerve irritations by infectious agents, chemicals, tumors, and trauma. Consideration to the aforementioned may prove lifesaving in some cases.

In the case reported here a clinical diagnosis of herpes zoster was made at the time of consultation, and he was returned to his referring physician with a recommendation for x-ray therapy. The initial clinical examination of the patient was directed primarily to a determination of the possibility of an infectious etiology or nerve irritation by tumefactions. The latter was ruled out by x-ray examination. The cause of the herpes zoster was then assumed to be primary in origin, that is, infectious (viral). In retrospect, the "heat rash" observed upon the patient's back and chest at the time of consultation was most likely an early transitory leukemid manifestation and thus should have suggested systemic investigation. The rash, however, was not assigned much importance in the illness, since the patient's past history revealed multiple episodes in which a rash followed exertion. Without question, a complete blood count taken at the time of consultation might have indicated the presence of leukemia, and thus antileukemic treatment could have been instituted at that time. I am unable at this time to recall the reason that a complete blood count was not included as part of the consultation. It should be pointed out that normally all patients examined in consultation at the department of oral surgery had a routine complete blood count and a urinalysis in addition to any specific tests required. The delay in establishing the diagnosis of leukemia was further complicated by the inability to get the patient to return for routine posttreatment checkups. Fortunately, some three months after the completion of x-ray therapy the patient was re-examined at the insistence of his family. Gross clinical symptoms suggestive of leukemia were present, and a complete blood study verified the clinical impression.

This case has been reported because of the interesting fact that oral and facial herpes zoster was the primary manifestation of a previously undiagnosed lymphatic leukemia and also to reiterate the necessity of routinely obtaining a complete blood count on every patient with a disease of possible systemic origin.

REFERENCES

1. Burket, Lester W.: Oral Medicine, ed. 5, Philadelphia, 1946, J. B. Lippincott Company, pp. 147, 151-153.
2. Cecil, Russell L., and Loeb, Robert F.: A Textbook of Medicine, ed. 9, Philadelphia, 1955, W. B. Saunders Company, pp. 29, 30.
3. Rosenow, E. C., and Oftedal, S.: The Etiology and Experimental Production of Herpes Zoster, J. A. M. A. 64: 1968, 1915.
4. McEwen, F. L: The Association of Herpes Zoster With Varicella, Arch Dermat. & Syph. 2: 205, 1920.
5. Feller, F., and Schnabel, T. F.: Concurrent Herpes Zoster and Chickenpox, Arch. Int. Med. 83: 502-504, 1949.
6. Thoma, Kurt H.: Oral Pathology, ed. 3, St. Louis, 1950, The C. V. Mosby Company, pp. 1174-1176.
7. Wilson, S. A. K., and Bruce, A. N.: Neurology, ed. 2, Baltimore, 1955, Williams & Wilkins Company, p. 784.
8. Weinberger, W.: Herpes zoster nach Zahnextraktion, Ztschr. f. stomatol. 31: 441, 1933.
9. Head, H.: In Allbut, T. C., and Rolleston, H. (editors): System of Medicine, ed. 2, London, 1910, Macmillan & Co., Ltd., pp. 7, 480.
10. Berggreen, P., and Schüler, E. G.: Zur Kenntnis der Lokalisation des Herpes zoster, Dermat. Wchnschr. 106: 216, 1938.
11. Dennis, F. L.: Herpes Zoster Oticus; a Case With Involvement of the 5th, 7th, 8th, and 9th Cranial Nerves With Complete Vestibular Examination, Laryngoscope 35: 665, 1925.
12. Campbell, R. M.: Bilateral Herpes of the Trigeminal, Lancet 1: 1066-1067, 1936.
13. Boyd, William: A Textbook of Pathology, ed. 5, Philadelphia, 1950, Lea & Febiger, p. 908.
14. Hoak, Frank C.: Personal Communication.
15. Sheehan, George L.: Personal Communication.

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RECURRENT APHTHOUS ULCERATIONS AND RECURRENT HERPES LABIALIS IN A PROFESSIONAL SCHOOL STUDENT POPULATION

III. Oral Examinations

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INTRODUCTION

RECURRENT aphthous ulcerations, also known as "canker sores," appear as painful ulcerations on the oral mucous membranes. Females appear to be more susceptible than males, as reported in two independent studies.[1, 2] Of 1,788 medical, dental, veterinary, dental hygiene, and nursing students in the University of Pennsylvania area, 55 per cent reported a positive history of this condition. Of the students with positive histories of aphthous ulcers, 2 per cent reported recurrences at daily or weekly intervals, 12 per cent had them at monthly intervals, 49 per cent indicated episodes of intraoral ulcerations at intervals of from two to eleven months, and 34 per cent reported annual or less frequent ulcerations.[1]

Analyses of medical history data from 64 per cent of this population revealed that the aphthous-positive group reported significantly more emotional problems than students who denied having this condition and that the extent of this factor was related to the severity of the disease, as interpreted in frequency of intraoral ulceration. In addition, relationships between recurrent aphthous ulcerations and eye, ear, nose, and throat systems, as well as infectious diseases, allergy, cardiorespiratory, gastrointestinal, neurological, and emotional categories for male students and eye, ear, nose, and throat, neurological, and emotional categories for female students were seen.[3]

Although considerable attention has been devoted to the epidemiological investigation of oral disease in the past few years, these studies have, for most part, been concentrated exclusively on dental diseases. Thus, whereas epidemiological tools for the investigation of caries and periodontal diseases have been

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developed, no attempts have been made to document the prevalence of other soft-tissue findings, such as "white lesions," aphthous ulcerations, pericoronitis, petechial hemorrhages within the mucosa, geographic tongue, fever blisters, and other not readily classifiable inflammations within the oral soft tissues. In 1955, Stahl and Morris[4] recorded the prevalence of "white lesions" and gingival disease among army personnel. They found that 11.6 per cent of servicemen in the 17- to 19-year age group and 6.0 per cent in the 20- to 29-year age group demonstrated "white lesions" on oral examination.[4] Analyses of questionnaire data regarding the frequency of various oral conditions submitted to the *New York State Dental Journal* revealed that 1.72 per cent of 5,591 patients reported on by seventy-six dentists who replied to the questionnaire had keratoses, leukoplakia, or lichen planus, three conditions that frequently cause the appearance of "white lesions."[5]

It was felt that thorough examination of the oral soft tissues of professional school students who reported a history of recurrent aphthous ulcerations and who indicated recurrences of lesions at intervals of up to one year might contribute to an increased understanding of the frequency with which these findings appear. In addition, it was felt that such examinations might provide additional insight into the problem of recurrent aphthous ulcerations of the mouth, as no such examinations have been reported except in individual patients.

MATERIALS AND METHODS

The student population at the University of Pennsylvania schools of medicine, dentistry, veterinary medicine, dental hygiene, and graduate nursing, and the University of Pennsylvania Hospital Nursing School, the Philadelphia General Hospital School of Nursing, and the Presbyterian Hospital School of Nursing completed a questionnaire regarding their history of recurrent aphthous ulcerations in the mouth.[1, 3] Students who reported recurrences of aphthous lesions in the mouth at daily, weekly, monthly, and greater than annual intervals were selected from the entire population and were requested to have dental examinations by members of the study team (I.I.S., A.L.M., and R.T.D.). Arrangements were made with the individual student through the school offices, and appointments were made to coordinate with the student's schedule.

Examinations were performed at the respective schools involved. A portable Burton dental lamp, mirrors, sponges, finger cots, and a standard examination form were used throughout. Two dental mirrors were used for each oral examination. Examiner comparability was checked by independent evaluation of the same students by all examiners, with subsequent agreement on broad clinical diagnostic criteria. Examinations were recorded in shorthand and reviewed at a subsequent date.

The sequence of each oral examination was determined by a protocol, and negative findings were recorded for each category before the next area was examined. Thus, all examinations were of comparable thoroughness in that all indicated areas were observed and palpated. The examination sequence was as follows: The patient was first interviewed in order that data received on

questionnaire forms might be checked.[1, 3] The head and neck were carefully observed for swellings, color changes in the skin, and asymmetry. The neck and submandibular structures were palpated to determine the presence of palpable lymph nodes. Temporomandibular joint motions were made with the examiner's fingers on the external condylar regions. Following this, the external aspects of the upper and lower lips were examined and palpated. Crusting, drying, fissuring, and specific lesions were described. The mucosal aspect of the upper and lower lips, including the labial vestibules, was reflected and examined by observation and palpation. Color, texture, masses, and specific lesions were noted and described. The buccal mucosa was likewise reflected and examined by observation and palpation. Pressure was applied to the parotid ducts bilaterally, and the resultant salivary flow was observed. Following examination and palpation of the anterior third of the tongue, a gauze sponge was applied, and the tongue was extended from the mouth. The floor of the mouth was carefully examined. The lateral margins, the ventral surface, and the base of the tongue were thus examined. Following this, the patient protruded the tongue, and the base and lateral margins of the tongue were palpated bimanually. Pressure was applied bilaterally on the submaxillary salivary glands and ducts, and the resultant flow from the duct orifices was examined. The soft and hard palates were inspected, and the oral pharynx was examined while the patient was gagged briefly with the dental mirror. The gingivae were carefully inspected, but only gross periodontal inflammation associated with specific bleeding from the gingival tissues and acute pericoronitis surrounding erupting third molars were recorded. Examinations of teeth were limited to a recording of the number of missing, filled, and carious teeth in the mouth. Specific inflammations, petechial hemorrhages, and raised, white, lined plaques, patches, and streaks were given particular attention. Specific pathologic formations were individually described. The nature and quality of lingual papillae, the presence or absence of enlarged tonsils, and the over-all appearances of the oral tissues were documented.

Following each examination, the student was instructed in the maintenance of keysort diary forms and was requested to document each lesion by location. The results of this study will be reported on at a later date.[6]

RESULTS

Of the 1,788 students in the original population, a total of 610 reported recurrences of aphthous ulcerations in the mouth at daily, weekly, monthly, or more frequently than annual intervals.[1] Of this group, 300 students reported for examination (49.2 per cent). Table I shows the distribution of students examined by severity of disease as compared with the original population.

Evidence of recurrent aphthous ulcerations, as characterized by the finding of a typical morphologic lesion within the oral tissues, was seen in 46 per cent of the students examined. Whereas 91 per cent of the students reporting daily or weekly ulcerations were found to have active aphthous lesions, 73 per cent

TABLE I. DISTRIBUTION OF STUDENTS WITH RECURRENT APHTHOUS ULCERATIONS IN THE POPULATION THAT RECEIVED ORAL EXAMINATIONS AND PREVALENCE OF RECURRENT APHTHOUS ULCERS (RAU) FOUND ON EXAMINATION BY SEVERITY GROUP

SEVERITY GROUP	TOTAL ORIGINAL POPULATION	TOTAL EXAMINED POPULATION	PER CENT EXAMINED	TOTAL WITH R.A.U.	PER CENT WITH R.A.U.
Daily and weekly	19	11	57.9	10	90.9
Monthly	111	60	54.0	44	73.3
Two to eleven ulcers per year	480	229	47.7	83	36.2
Total	610	300	49.2	137	45.7

of the students reporting monthly episodes and 36 per cent of the students reporting lesions at intervals of from two to eleven months demonstrated aphthous ulcerations on oral examination (Table I).

The distribution of aphthous lesions in the mouth by location is seen in Fig. 1.

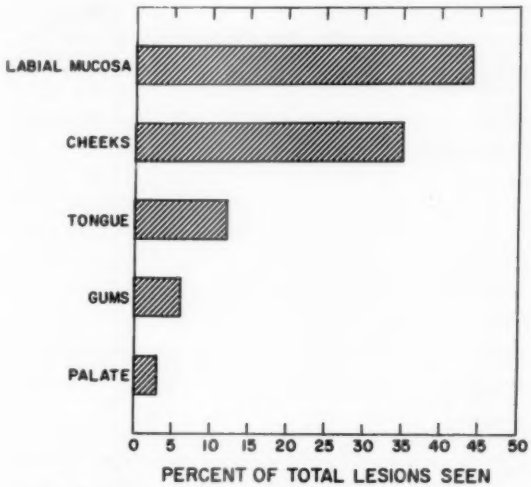

Fig. 1.—Frequency of recurrent aphthous ulcerations found on oral examination by location in the mouth.

Other findings on soft-tissue examination of the mouth are indicated in Fig. 2. Of the students examined, 54.5 per cent demonstrated soft-tissue changes other than aphthous ulcerations or gingivitis. Palpable lymph nodes were found in the neck or submandibular regions of 167 students (55.5 per cent); "white lesions" were seen in ninety-seven students (32.2 per cent); gingivitis was noted in 100 students (33.3 per cent); and forty-three students (14.3 per cent) had subacute to chronic pericoronitis surrounding an erupting third molar at the time of examination. Petechial hemorrhages on the oral mucous membranes were observed in twenty-three students (7.6 per cent), geographic tongue was seen in eight (2.6 per cent), and acute Vincent's infection (acute ulcerative necrotizing gingivitis) was noted in two (0.7 per cent). Fordyce spots were

noted on the buccal and labial mucosa of forty-four students (14.6 per cent), and recurrent herpes labialis (fever blisters) was found in fifteen students (5.0 per cent). Nonspecific inflammations of the oral mucous membranes were recorded by location in the mouth. These did not appear to be diagnostic for any specific disease, and they were only described and recorded by location.

Comparison of results recorded by individual examiners revealed close correlations as regards frequency distributions for each finding in various groups. No significant differences within the population studied were observed to be related to age, sex, school, class, marital status, race, and religion. Analyses of oral soft tissue findings for aphthous severity groups revealed no differences.

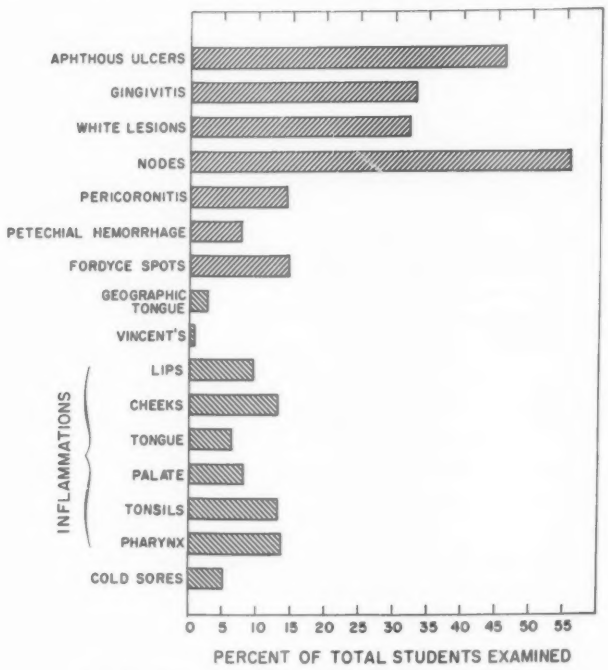

Fig. 2.—Frequency of oral soft-tissue findings on oral examinations.

DISCUSSION

Since the results of specific pathologic examinations of biopsy material removed from students demonstrating "white lesions" on the oral mucous membranes are not available, no specific diagnostic insight can be derived from these data. These lesions could have represented leukoplakia, lichen planus, responses to trauma or lip- and cheek-biting, or other such diseases. Nevertheless, it would appear that these findings are more common than one might expect from a population of this age, sex, and background.

That 46 per cent of these students demonstrated evidence of active aphthous ulcerations in the mouth at the time of examination supports our previous findings that the experience of recurrent aphthous ulcerations in a student population can be determined from history alone.[1, 3] This is further borne out by the fact that the students examined in each severity category demonstrated aphthous lesions according to their history of disease, that is those with more frequent episodes of oral ulcerations demonstrated significantly more lesions on oral examination than the groups with a history of fewer recurrences. Nevertheless, students with active aphthous ulcerations at the time of examination did not demonstrate any increased frequency of other oral soft-tissue diseases. Since no control group (that is, students with a negative history of recurrent aphthous ulcerations) was examined, however, these findings might represent abnormal tissue responses related to recurrent aphthous ulcerations. Certainly this study should be repeated with the inclusion of an aphthous-negative group.

Another finding that tends to support the population statistics for both recurrent aphthous ulcerations and recurrent herpes labialis, as previously reported, was the finding that 5 per cent of students examined demonstrated typical fever blisters or cold sores.[1, 3] Review of these students' charts revealed that all had indicated frequent episodes of both diseases by history. One hundred fourteen students (38.0 per cent) examined also gave a history of recurrent herpes labialis. Of these, only 3 per cent reported monthly or more frequent episodes of disease. All nine students examined within this group demonstrated herpetic vesicles on the lips, whereas the other six students in whom this disease was found reported less than monthly recurrences.

It is interesting that the location of aphthous ulcerations by history showed some differences when compared with the actual location of lesions on oral examination. Analyses of results of medical history forms indicated the following ranking of lesions by location: (1) lips (29.8 per cent), (2) cheeks (29.8 per cent), (3) gingiva (17.1 per cent), (4) tongue (11.6 per cent), and (5) palate (4.4 per cent). Thus, oral examination disclosed more lesions on the lips and tongue and fewer on the gingiva than indicated in the histories. Examination of these results by age, sex, and severity of disease revealed no differences, but the variations within each group were considerable. No differences between schools and classes were observed except for the dental students, in whom very close correlations were noted between location of lesions by history and on examination. Perhaps these discrepancies represent a vague understanding by nondental students of the geographic differences of these locations within the mouth.

Since all examinations were performed in the winter months (January and February), tonsillar and pharyngeal inflammations associated with lymph node enlargement were common. The prevalence of gingivitis, pericoronitis, Fordyce spots, geographic tongue, and Vincent's infection might well reflect the situation in any group of 300 persons of this age and sex distribution; however, it would appear that the high prevalence of white lesions on the oral mucous membranes noted in this group is unusual and deserves further investigation.

SUMMARY AND CONCLUSION

Three hundred students attending the University of Pennsylvania schools of medicine, dentistry, veterinary medicine, dental hygiene, and nursing, and the University of Pennsylvania Hospital School of Nursing, the Philadelphia General Hospital School of Nursing, and the Presbyterian Hospital School of Nursing who reported recurrences of aphthous ulcerations in the mouth at daily, weekly, monthly, and less frequent intervals not exceeding one year, were subjected to thorough oral soft-tissue examinations. Aphthous ulcerations were observed in 46 per cent of the group; 91 per cent of the students who reported daily or weekly recurrences, 73 per cent of those who reported monthly recurrences, and 36 per cent of those who reported from two to eleven recurrences of aphthous ulcerations each year had evidence of active disease at the time of examination. In addition, "white lesions," gingivitis, cervical and submaxillary lymphadenopathy, pericoronitis around erupting third molars, petechial hemorrhages within the oral mucosa, Fordyce spots, geographic tongue, and other nonspecific soft-tissue changes were described. The prevalence of "white lesions" appeared to be excessive in the population studied (32.2 per cent).

The authors would like to thank Mr. Carl L. White, Epidemiology and Biometry Branch, National Institute of Dental Research, for his invaluable assistance in preparing the statistical analyses, and Mrs. Olivia Richardson, without whose diligent labors this work would not have been completed.

REFERENCES

1. Ship, I. I., Morris, A. L., Durocher, R. T., and Burket, L. W.: Recurrent Aphthous Ulcerations and Recurrent Herpes Labialis in a Professional School Student Population. I. Experience, ORAL SURG., ORAL MED. & ORAL PATH. 13: 1191-1202, 1960.
2. Sircus, W., Church, R., and Kelleher, J.: Recurrent Aphthous Ulceration of the mouth, Quart. J. Med. 26: 235-250, 1957.
3. Ship, L. I., Morris, A. L., Durocher, R. T., and Burket, L. W.: Recurrent Aphthous Ulcerations and Recurrent Herpes Labialis in a Professional School Student Population. II. Medical History, ORAL SURG., ORAL MED. & ORAL PATH. 13: 1317-1329, 1960.
4. Stahl, S. S., and Morris, A. L.: Oral Health Conditions Among Army Personnel at the Army Engineering Center, J. Periodont. 26: 180-185, 1955.
5. Kutscher, A. H., Zegarelli, E. V., and Mercadante, J.: Incidence or Oral Diseases: Results of Cooperative Research Survey IV, New York State Dent. J. 25: 135-136, 1959.
6. Ship, I. I., Morris, A. L., Durocher, R. T., and Burket, L. W.: Recurrent Aphthous Ulcerations in a Professional School Student Population. IV. Twelve-Month Study of Natural Disease Patterns. (In preparation.)

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OSTEOPOROSIS OF THE JAWS ASSOCIATED WITH HYPERCORTISONISM

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THE current clinical term "hypercortisonism" connotes not merely an excess of cortisone but also the effects of the administration of excessive amounts of other related effective adrenocortical hormones. In other words, "hypercortisonism" refers to the effects of excessive stimulation of the adrenal cortex resulting from the administration of a corticotropin.[3] One of the undesirable side effects of excessive stimulation of the adrenal cortex by cortisone or one of the newer synthetic analogues is osteoporosis or rarefaction of bone, particularly the pelvis and vertebral column.[4, 5] Zegarelli[6] has reproduced roentgenograms in which this type of osteoporosis is demonstrated in the jaws. In some instances the osteoporosis may be so severe that pathologic fracture of bones results.[2]

According to one paper,[1] cortisone affects the metabolism of bone at three sites: (1) the bone itself, (2) the gastrointestinal tract, and (3) the renal tubules. The changes in bone which ensue are attributed to decreased formation of osteoid tissue, the proteinaceous matrix in which bone salts are deposited. Under certain circumstances corticosteroids also inhibit the absorption of calcium from the gastrointestinal tract, and this is evidenced by an increased fecal excretion of calcium. It also has been found that administration of cortisone may increase the urinary excretion of phosphate by depressing reabsorption of phosphate by the renal tubules.

The histologic aspects of bone changes in the presence of hypercortisonism are different from those which are seen in hyperparathyroidism in that there are

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no cystic changes and no formation of giant-cell tumor which is so characteristic of the latter condition. The bone spicules are reduced in number and thickness; therefore, the roentgenologic appearance is one that may be marked roentgenolucent.

In the jaws the osteoporosis is manifested roentgenologically by fine and indistinct trabeculae, thinning or obliteration of the cortex, and partial or complete obliteration of the lamina dura. These features are shown in Fig. 1, which was selected from a number of dental roentgenograms made for a 45-year-old man who had used various corticosteroids without interruption for eight years. Examination of the head and the lumbar portion of the spinal column also revealed osteoporosis. The osteoporosis of the jaws could not be

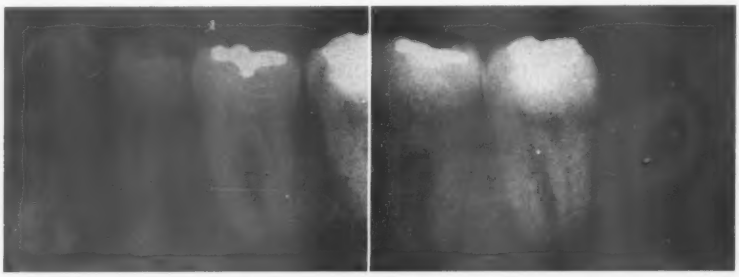

Fig. 1.—Osteoporosis of the mandible associated with hypercortisonism as evidenced by fine, indistinct trabeculae and absence of the lamina dura.

attributed to disuse, since all the teeth anterior to the third molars were present and were in normal occlusion and normal function. In the dental roentgenogram the appearance of the osteoporosis in the presence of hypercortisonism is similar to that which is sometimes associated with Cushing's syndrome, in that the bone of the jaws has a uniform roentgenolucent quality and the lamina dura may be completely obliterated. In osteoporosis associated with senility, postmenopause, or disuse, the lamina dura may become very thin but is discernible in the roentgenogram.

REFERENCES

1. [Anderson, J. T.]: Cortisone Effects on Bone Mineral Metabolism, Nutrition Rev. 16: 247-248, 1958.
2. Curtiss, P. H., Jr., Clark, W. S., and Herndon, C. H.: Vertebral Fractures Resulting From Prolonged Cortisone and Corticotropin Therapy, J. A. M. A. 156: 467-469, 1954.
3. Slocumb, C. H., Polley, H. F., Ward, L. E., and Hench, P. S.: Diagnosis, Treatment and Prevention of Chronic Hypercortisonism in Patients With Rheumatoid Arthritis, Ann. Int. Med. 46: 86-101, 1957.
4. Soffer, L. J.: Diseases of the Endocrine Glands, ed. 2, Philadelphia, 1956, Lea & Febiger, pp. 365-368.
5. Turner, C. D.: General Endocrinology, ed. 2, Philadelphia, 1955, W. B. Saunders Company, p. 176.
6. Zegarelli, E. V.: Dental Roentgenology. In Golden, Ross: Diagnostic Roentgenology, vol. 3, Baltimore, 1959, The Williams & Wilkins Company, pp. 76-77.

RELATIONSHIP OF THREE-HOUR BLOOD GLUCOSE TO ORAL ROENTGENOGRAPHIC FINDINGS

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INTRODUCTION

A SURVEY of the literature shows that many investigators have a tendency to associate the roentgenographic findings of alveolar bone loss and periodontal widening with the diabetic patient. There is implication of a linear relationship between these two findings and the diabetic state. In other words, the more pronounced the diabetic state (the higher the blood glucose level), the more severe is the alveolar bone loss and/or periodontal widening.

One report[1] has already analyzed the relationship of oral roentgenographic findings with the *glucose-tolerance patterns.* The evidence from that study indicates that there is a *linear* relationship between the glucose-tolerance pattern and the presence of alveolar bone loss and periodontal widening. Two other reports[2, 3] have shown the relationship of oral roentgenographic findings with the *fasting* and *two-hour* glucose levels. The results of these studies point to a *parabolic* relationship between the fasting and two-hour glucose levels and the presence of alveolar bone loss and periodontal widening.

The present report will analyze the relationship of the two roentgenographic signs (alveolar bone loss and periodontal widening) to the *three-hour* blood true glucose in a group of 100 routine dental patients.

REVIEW OF THE LITERATURE

Published material is available regarding the relationship between diabetes mellitus and (1) alveolar bone loss and (2) periodontal widening.

Alveolar Bone Loss.—Many investigators have reported this finding as being associated with the diabetic picture.[4-10] One investigator[11] observed bone loss in one out of every two of a group of diabetic children. Two teams,[12, 13] however, claim no alveolar bone loss. Sheridan and associates[14] observed normal

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Editor's Note: This paper may appear to relate more closely to other disciplines than to oral roentgenology. It was accepted for publication because of its quality and in an effort to suggest that this type of investigative effort could profitably be performed by oral roentgenologists as well as by those having a primary interest in biochemical endeavor.

alveolar bone in 24 per cent of patients with normal glucose-tolerance patterns. They further noted that patients with increased or decreased glucose tolerance showed a greater incidence of alveolar bone loss and that most of the diabetic patients demonstrated considerable bone loss in contrast to other groups of patients who had more physiologic glucose-tolerance patterns.

Periodontal Widening.—Rutledge[11] observed this finding in fourteen out of twenty juvenile diabetic patients. The observations of Rudy and Cohen[13] in this respect are in agreement with those of Rutledge. However, Ziskin and colleagues[15] could not demonstrate periodontal widening in their series of cases. Sheridan and his co-workers[14] reported that in patients with decreased glucose-tolerance patterns (persons who would commonly be called diabetic) there was a 62 per cent incidence of periodontal widening. These investigators also observed that the occurrence of this finding decreased with an increase in sugar tolerance.

METHOD OF INVESTIGATION

One hundred dental patients were studied in the Section on Oral Medicine at the University of Alabama School of Dentistry. As far as possible, the subjects were selected at random. Table I shows the age distribution of the group. It can be observed that the pattern approaches a typical unimodal curve. Included in this study were twenty-two male patients and seventy-eight female patients.

TABLE I. AGE DISTRIBUTION

AGE GROUP	TOTAL NUMBER OF PATIENTS
0- 9	1
10-19	9
20-29	19
30-39	27
40-49	19
50-59	17
60-69	8
Total	100

The roentgenograms of each patient were studied for possible alveolar bone loss and periodontal widening. Alveolar bone loss was scored on a seven-point scoring system where 0 was the score given for a normal septal crest; a score of 1 was assigned for minimal alveolar bone loss, 2 for minimal to moderate alveolar bone loss, 3 for one-half the alveolar support missing, 4 for moderate to severe alveolar bone loss, 5 for no alveolar support, and 6 for edentulous cases. Periodontal widening was scored on a four-point scoring system where 0 was the score given for no marginal widening; a score of 1 was assigned for minimal marginal widening, 2 for severe marginal widening, and 3 for edentulous cases.

A true glucose-tolerance test was performed on each patient according to the methods of Somogyi[16, 17] and Nelson.[18] No preparatory diet was recommended other than complete fasting for twelve hours prior to the laboratory

examination. Complete details regarding the method of investigation are available in the article by Sheridan and associates.[14] For this study, only the information derived from the three-hour blood glucose measurements was utilized.

RESULTS

The three-hour blood glucose determination will be considered in the light of (1) single roentgenographic signs and (2) a combination of the two signs.

Single Signs

An analysis of the data will be offered for (1) alveolar bone loss and (2) periodontal widening.

Alveolar Bone Loss.—This roentgenographic sign will be discussed in terms of (1) mean scores, (2) coefficients of variation, and (3) statistical significance.

The scores for edentulous patients were included in the analysis, since this condition would be an end result of alveolar bone loss. Although loss of teeth can be due to other causes, it is felt that the exclusion of these cases would be too discriminatory against those cases in which tooth loss was due to alveolar bone loss. The available data do not permit the selection of those cases in which loss of teeth was accompanied by severe alveolar bone loss.

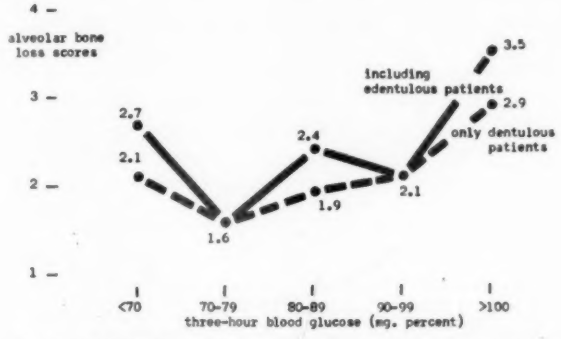

Fig. 1.—Relationship of three-hour blood glucose to alveolar bone loss.

Mean scores: Fig. 1 shows that alveolar bone loss was least in those patients with a three-hour blood glucose of 70 to 79 mg. per cent. There was an increase in the scores for patients in the $>$ 70, 80 to 89, and 90 to 99 mg. per cent groups. There was a marked increase for patients in the $>$ 100 mg. per cent group. Fig. 1 also shows a comparison with the mean scores obtained from dentulous cases alone. These scores show how the values for this roentgenographic sign can be affected by the exclusion of patients who have lost all their teeth.

Coefficients of variation: Table II reveals that the lowest coefficients of variation are exhibited by the $>$ 100, 90 to 99, and $<$ 70 mg. per cent groups. The conclusion to be drawn is that the individual scores seem to cluster about the mean more in those patients with hyperglycemia and hypoglycemia.

TABLE II. RELATIONSHIP OF THREE-HOUR BLOOD GLUCOSE TO ALVEOLAR BONE LOSS

	BLOOD GLUCOSE GROUPS (MG. PER CENT)				
	< 70	70 TO 79	80 TO 89	90 TO 99	> 100
Mean alveolar bone loss score	2.7	1.6	2.4	2.1	3.5
Standard deviation	1.8	1.3	1.7	1.4	1.8
Coefficient of variation	67	81	71	67	51
Mean difference	1.1	0.8	0.3	1.4	
Standard error of difference	0.5	0.6	0.6	0.6	
Observed t value	2.20	1.33	0.50	2.33	
Degrees of freedom	52	30	27	24	
P	< .05	> .1	.5	< .05	

Statistical significance: Table II shows the probability values obtained when the blood glucose groups were compared to similar groups that were greater or lesser by 10 mg. per cent. Significant differences are shown between the < 70 and 70 to 79 mg. per cent groups and the 90 to 99 and > 100 mg. per cent groups. This is evidenced by probability values of < .05. The P value was also checked for all the groups. No statistically significant differences were shown except when the 70 to 79 and > 100 mg. per cent groups were compared. The P value obtained from this comparison was < .005.

Periodontal Widening.—This roentgenographic sign will be discussed in terms of (1) mean scores, (2) coefficients of variation, and (3) statistical significance.

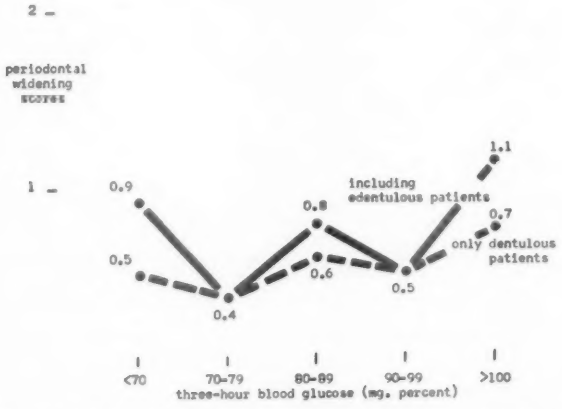

Fig. 2.—Relationship of three-hour blood glucose to periodontal widening.

Mean scores: Fig. 2 shows the mean scores of all patients (dentulous and edentulous) and the mean values obtained when edentulous patients were excluded. An examination of these scores shows the effect of excluding the edentulous patients. Patients without teeth would not be counted and thus lower the mean score of the respective blood glucose groups. The lowest mean score

for this roentgenographic sign is shown by the 70 to 79 mg. per cent group, and basically this condition increases as the three-hour blood glucose rises above or falls below this small glucose range.

Coefficients of variation: Table III shows that the lowest coefficient of variation is obtained in the > 100 mg. per cent group, although others demonstrate almost the same coefficient of variation. In other words, the individual scores more closely cluster about the mean in these groups.

TABLE III. RELATIONSHIP OF THREE-HOUR BLOOD GLUCOSE TO PERIODONTAL WIDENING

	BLOOD GLUCOSE GROUPS (MG. PER CENT)				
	< 70	70 TO 79	80 TO 89	90 TO 99	> 100
Mean periodontal widening score	0.9	0.4	0.8	0.5	1.1
Standard deviation	1.1	0.4	0.9	0.5	1.0
Coefficient of variation	122	100	112	100	91
Mean difference	0.5	0.4	0.3	0.6	
Standard error of difference	0.2	0.2	0.3	0.4	
Observed t value	2.50	2.00	1.00	1.50	
Degrees of freedom	52	30	27	24	
P	<.025	>.05	>.2	>.1	

Statistical significance: From Table III it can be seen that there are no statistically significant differences between any of the blood glucose groups when compared to similar groups that are greater or lesser by 10 mg. per cent of blood glucose, except between the < 70 and 70 to 79 mg. per cent groups. This is evidenced by a probability value of < .025. The P value was also checked for all the groups. No statistically significant differences were shown except when the 70 to 79 and > 100 mg. per cent groups were compared. A P value of < .05 was obtained when the scores of these two groups were analyzed.

Combination of the Two Roentgenographic Signs

An analysis of the data will be offered for alveolar bone loss and periodontal widening. The combination of these two signs will be discussed in terms of: (1) mean scores, (2) coefficients of variation, and (3) statistical significance. There exists a difference in the scoring systems for these two signs, alveolar bone loss being measured on a seven-point scale and periodontal widening on a four-point scale. In order to equate these scores, the periodontal scores were doubled so that a value could be placed on the combination of these two roentgenographic signs.

Mean scores: Fig. 3 shows the relationship between three-hour blood glucose levels and the presence of these two roentgenographic signs. The mean scores are lowest in the 70 to 79 mg. per cent range, and basically the scores increase in the higher and lower blood glucose groups.

Coefficients of variation: Table IV shows that the lowest coefficients of variation occur in the 70 to 79 and > 100 mg. per cent blood glucose groups. This indicates that the individual scores more closely cluster about the mean in these two blood glucose groups.

Statistical significance: Table IV shows that when the three-hour blood glucose groups are compared with similar groups differing by 10 mg. per cent of blood glucose there are significant differences between the > 70 and 70 to 79 mg. per cent groups and the 90 to 99 and > 100 mg. per cent groups. Both of these comparisons show a P value of < .025. A comparison of each group with all others showed no statistically significant differences except between the 70 to 79 and > 100 mg. per cent groups. The P value here was < .005.

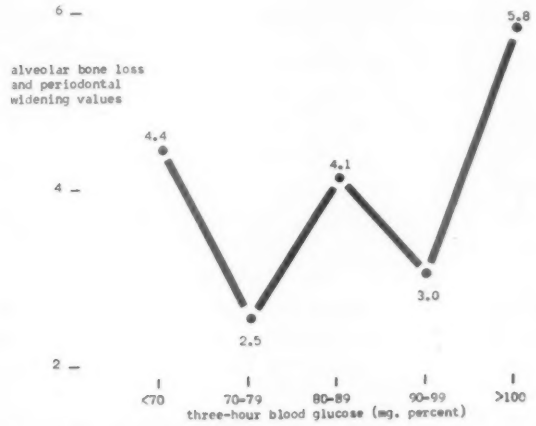

Fig. 3.—Relationship of three-hour blood glucose to alveolar bone loss and periodontal widening (100 routine dental patients).

DISCUSSION

The data will be discussed in terms of (1) mean scores, (2) coefficients of variation, and (3) significant probability values. Because of the close correlation between age versus glucose levels and roentgenographic signs versus age, the age factor will be analyzed in terms of blood glucose and roentgenographic signs.

TABLE IV. RELATIONSHIP OF THREE-HOUR BLOOD GLUCOSE TO ALVEOLAR BONE LOSS AND PERIODONTAL WIDENING

	BLOOD GLUCOSE GROUPS (MG. PER CENT)				
	< 70	70 TO 79	80 TO 89	90 TO 99	> 100
Mean alveolar bone loss and periodontal widening values	4.4	2.5	4.1	3.0	5.8
Standard deviation	3.9	1.6	3.6	2.3	3.7
Coefficient of variation	89	64	88	77	64
Mean difference		1.9	1.6	1.1	2.8
Standard error of difference		0.7	1.0	1.1	1.1
Observed t value		2.64	1.63	0.96	2.46
Degrees of freedom		52	30	27	24
P		<.025	>.1	>.2	<.025

Mean scores: An analysis of the mean score values for the two roentgenographic signs independently and in combination tends to indicate that there is a range of blood glucose normality for a particular roentgenographic sign or group of signs (Table V) in the general range of 70 to 79 mg. per cent. This information also suggests that three-hour blood glucose levels above and below these narrow ranges are likely to be associated with positive roentgenographic findings.

TABLE V. COMPARISON OF MEAN SCORES

	BLOOD GLUCOSE GROUPS (MG. PER CENT)				
	< 70	70 TO 79	80 TO 89	90 TO 99	> 100
Alveolar bone loss	2.7	1.6	2.4	2.1	3.5
Periodontal widening	0.9	0.4	0.8	0.5	1.1
Alveolar bone loss and periodontal widening	4.4	2.5	4.1	3.0	5.8

Coefficients of variation: Generally speaking, the lower the coefficient of variation, the more the individual scores cluster about the mean. Thus, a coefficient of variation of 0 is the ultimate in that it signifies that all subjects provide scores exactly like the mean.

Table VI summarizes the coefficients of variation with respect to the three-hour blood glucose groups and the single and combined roentgenographic signs. It can be seen that the individual scores cluster more closely about the mean in the > 100 mg. per cent group.

TABLE VI. COMPARISON OF COEFFICIENTS OF VARIATION

	BLOOD GLUCOSE GROUPS (MG. PER CENT)				
	< 70	70 TO 79	80 TO 89	90 TO 99	> 100
Alveolar bone loss	67	81	71	67	51
Periodontal widening	122	100	112	100	91
Alveolar bone loss and periodontal widening	89	64	88	77	64

Significant probability factors: Table VII gives a tabulation of the significant probability findings for the two single and combined roentgenographic signs. It is noteworthy that patients with three-hour blood glucose levels of < 70 and > 100 mg. per cent show significant differences in the amount of

TABLE VII. COMPARISON OF SIGNIFICANT PROBABILITY VALUES

	RELATIONSHIP OF THREE-HOUR BLOOD GLUCOSE GROUPS (MG. PER CENT)	P VALUE
Alveolar bone loss	< 70 with 70-79	< .05
	90-99 with > 100	< .05
	70-79 with > 100	< .005
Periodontal widening	< 70 with 70-79	< .025
	70-79 with > 100	< .05
Combination of alveolar bone loss and periodontal widening	< 70 with 70-79	< .025
	90-99 with > 100	< .025
	70-79 with > 100	< .005

alveolar bone loss and periodontal widening when compared to those with levels of 70 to 79 mg. per cent. Also noteworthy is the fact that the significant differences between these blood glucose groups are maintained when the roentgenographic signs are combined. It is of interest to observe that basically the significant differences observed for single roentgenographic signs tend to become more significant when the signs are combined.

Age factor: There are some investigators who contend that with increasing age the normal blood glucose values increase. Certainly, the data from which this study is derived indicate that roentgenographic signs, in the main, occur predominantly in the older age groups (Table VIII). Hence, it was thought necessary to analyze the relationship between roentgenographic signs and blood glucose levels in a single age group.

TABLE VIII. PERCENTAGE FREQUENCY OF PATIENTS WITH ROENTGENOGRAPHIC SIGNS BY AGE GROUPS

AGE GROUP	ALVEOLAR BONE LOSS	PERIODONTAL WIDENING	ALVEOLAR BONE LOSS AND PERIODONTAL WIDENING
0- 9	0	0	0
10-19	44	22	11
20-29	78	39	39
30-39	96	52	52
40-49	100	74	74
50-59	100	50	50
60-69	100	86	86

Fig. 4 demonstrates the relationship of three-hour blood glucose to alveolar bone loss and periodontal widening in the nineteen patients between the ages of 40 and 49. An examination of Fig. 4 and a comparison between these data and the findings shown in Fig. 3 indicates that when the age factor is eliminated the mean scores become definitely parabolic. A comparison of Tables IV and IX

TABLE IX. RELATIONSHIP OF THREE-HOUR BLOOD GLUCOSE TO ALVEOLAR BONE LOSS AND PERIODONTAL WIDENING (40 TO 49-YEAR AGE GROUP)

	BLOOD GLUCOSE GROUPS (MG. PER CENT)				
	< 70	70 TO 79	80 TO 89	90 TO 99	> 100
Mean alveolar bone loss and periodontal widening values	5.5	4.0	3.8	4.7	7.7
Standard deviation	3.7	0.0	1.1	1.6	3.8
Coefficient of variation	67	0	29	34	49
Mean difference	1.5	0.2	0.9	3.0	
Standard error of difference	1.5	0.6	1.1	2.4	
Observed t value	1.00	0.33	0.82	1.25	
Degrees of freedom	7	5	5	4	
P	>.2	.5	>.4	>.2	

shows that the individual scores for the one age group cluster more closely about the mean than the scores for the entire group as judged by the coefficients of variation. No significant differences were observed when each blood glucose group was compared to any other group. It thus appears that age or some other factor operating with age may be involved in the relationship of the two roentgenographic signs and glucose metabolism.

The findings of this analysis, together with those observed when similar analyses were made of the *fasting* and *two-hour* blood glucose levels,[2, 3] point to a range of blood glucose normality as developed by the two roentgenographic signs. It is suggested that the normal fasting blood glucose is within the 70 to 79 mg. per cent range, the normal two-hour level is in the 80 to 89 mg. per cent group, and the normal three-hour blood glucose level is within the 70 to 79 mg. per cent range. This indicates that when the body is subjected to an intake of glucose the time needed for removal of this increase in glucose from the blood is in the vicinity of three hours. Findings from the two-hour blood glucose levels indicate that most of the glucose intake is removed from the blood within the first two hours. However, blood glucose measured at two hours is not quite as significant as measurements made three hours after an intake of glucose.

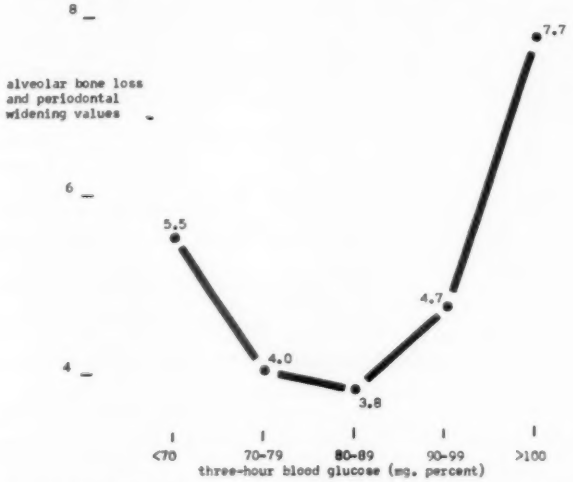

Fig. 4.—Relationship of three-hour blood glucose to alveolar bone loss and periodontal widening in patients 40 to 49 years of age.

SUMMARY

1. This report analyzes the relationship of two roentgenographic signs (alveolar bone loss and periodontal widening) to the three-hour blood true glucose level in a group of 100 routine dental patients.

2. An examination of the available data points out the need for evaluating edentulous patients as to the cause of loss of teeth.

3. An analysis of the mean score values for the two signs, independently and combined, shows that the lowest values were found in the 70 to 79 mg. per cent blood glucose groups.

4. A study of the individual scores (as expressed by the coefficients of variation) demonstrates that, in general, the individual values cluster more closely about the mean in the > 100 mg. per cent blood glucose group than in any of the other categories.

5. An analysis of the significant probability values shows that a significant difference exists between the 70 to 79 and > 100 mg. per cent three-hour blood glucose groups for both of the roentgenographic signs, singly and in combination.

6. The findings in this report suggest that the two roentgenographic signs (alveolar bone loss and periodontal widening) are associated with both hyper- and hypoglycemia.

7. The evidence derived in this report suggests that factors intimately associated with increasing age parallel increasing hyper- and hypoglycemia and the relationship of glucose imbalance with these two roentgenographic signs.

8. These data suggest that the range of three-hour blood glucose normality may be more narrow than usually accepted.

9. The findings of this study and other data obtained from analyses of the fasting[2] and two-hour[3] blood glucose levels point to ranges of blood glucose normality for determinations made at these times. Specifically, the fasting range is 70 to 79 mg. per cent, the two-hour range is 80 to 89 mg. per cent, and the three-hour range is again 70 to 79 mg. per cent of blood glucose.

REFERENCES

1. Cheraskin, E., and Manson-Hing, L.: The Normal Glucose-Tolerance Pattern: Development of Blood Glucose Normality by an Analysis of Oral Roentgenographic Findings, ORAL SURG., ORAL MED. & ORAL PATH. **13**: 819-835, 1960.
2. Manson-Hing, L., and Cheraskin, E.: The Relationship of Fasting Blood Glucose to Oral Roentgenographic Findings (in press).
3. Manson-Hing, L., and Cheraskin, E.: The Relationship of Two-Hour Blood Glucose to Oral Roentgenographic Findings (in press).
4. Rudy, A., and Cohen, M. M.: The Oral Aspects of Diabetes Mellitus, New England J. Med. **219**: 503-508, 1938.
5. Kaplan, N.: Oral Symptomatology of Diabetes Mellitus, Pennsylvania D. J. **41**: 50-51, 60-61, 1938.
6. Pollack, H., Person, P., and Knishkowy, E.: Diabetes Mellitus, Its Relation to Oral Pathosis. II. Oral Lesions Seen in Diabetes Mellitus, J. Periodont. **18**: 155-158, 1947.
7. Martinez, E.: Diabetes in Dental Practice, D. Absts. **1**: 178, 1956.
8. Stahl, S. S.: Roentgenologic and Bacteriologic Aspects of Periodontal Changes in Diabetes, J. Periodont. **19**: 130-132, 1948.
9. Golomb, I. M.: An Evaluation of the Relation of Diabetes Mellitus to Periodontal Disease, New York State Dent. J. **15**: 525-528, 1959.
10. Person, P.: Some Aspects of Bone Metabolism of the Maxilla and Mandible, New York State Dent. J. **23**: 165-170, 1957.
11. Rutledge, C. E.: Oral and Roentgenographic Aspects of the Teeth and Jaws of Juvenile Diabetics, J. Am. Dent. A. **27**: 1740-1750, 1940.
12. Lovestedt, S. A., and Austin, L. T.: Periodontoclasia in Diabetes Mellitus, J. Am. Dent. A. **30**: 273-275, 1943.
13. Rudy, A., and Cohen, M. M.: Oral Aspects of Diabetes Mellitus, J. Am. Dent. A. **29**: 523-534, 1942.
14. Sheridan, R. C., Jr., Cheraskin, E., Flynn, F. H., and Hutto, A. C.: Epidemiology of Diabetes Mellitus. II. A Study of 100 Dental Patients, J. Periodont. **30**: 298-323, 1959.
15. Ziskin, D. E., Siegel, E. H., and Loughlin, W. C.: Diabetes in Relation to Certain Oral and Systemic Problems. Part I. Clinical Study of Caries, Tooth Eruption, Gingival Changes, Growth Phenomena and Related Observations in Juveniles, J. D. Res. **23**: 317-331, 1944.
16. Somogyi, M.: A New Reagent for the Determination of Sugars, J. Biol. Chem. **160**: 61-68, 1945.
17. Somogyi, M.: Determination of Blood Sugar, J. Biol. Chem. **160**: 69-73, 1945.
18. Nelson, N.: A Photometric Adaptation of the Somogyi Method for the Determination of Blood Sugar, J. Biol. Chem. **153**: 375-380, 1944.

ROENTGENO-QUESTIONS

I often see a shadow which makes the upper portions of the crowns of the upper incisors appear more roentgenopaque than the incisal portions of the crowns. Is this caused by the extreme thinness of the incisal edges of some teeth?

There may be several reasons why this would occur. The incisal half of some teeth is extremely thin. This could cause the appearance described in the question. Also, the shadow of the upper lip may be superimposed on the upper half of the incisal crowns. In addition, with excessive vertical angulation, the shadow of the nose may be superimposed upon a portion of the crown, as well as upon the shadow of the lip. If excessive vertical angulation is used, the labial bone might be superimposed over the gingival third of the crown. A heavy palatal cingulum, particularly on central incisors, can also contribute to an increased roentgenopacity at the gingival third of the incisor tooth.

Sometimes the roots of the upper molars appear to project into the maxillary sinus with no lamina dura or floor of the sinus appearing about the apices. Why does this appear to occur?

In the absence of disease, the roots of the upper posterior teeth do not project into the maxillary sinus cavity without an osseous component (lamina dura) forming the alveolus. It frequently happens, however, that the lamina dura and the bony floor of the maxillary sinus appear to be one and the same structure. Frequently, the buccal and lingual roots of the upper posterior teeth are widely divergent and the maxillary sinus occupies space between these roots. On projection of the images onto the films, the third dimension, that of depth, is lost and all structures in the line of projection appear on the same place on the film. This often makes the roots appear to invade the sinus. If the films have sharp contrast (a tendency toward a predominance of blacks and whites with limited grays) or if the film density is excessive, the thin osseous structures around the root tips are "burned out," that is, destroyed by roentgenographic means. Teeth which show an absence of a lamina dura (whether projected into or superimposed on the sinus or totally divorced from the sinus) should be questioned and tested clinically for vitality.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

A Roentgenographic Artifact

THE accompanying series of x-ray films demonstrates the roentgenopacity of one type of antibiotic cone (Ilotycin) used in exodontia and oral surgery. The first film (Fig. 1) shows a retained root fragment. Two Ilotycin cones had

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Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

been placed in the socket before the first postoperative film was taken. This fact was overlooked when the film in Fig. 2 was viewed. It was deemed advisable to take two experimental films before re-entering the socket. Figs. 3 and 4 show the density of the Ilotycin cone per se and the density of the cone superimposed on a normal edentulous area. The last film in the series (Fig. 5) shows the surgical area postoperatively after the cones had dissolved.

W. J. Esser,
Sheboygan, Wis.

ORAL PATHOLOGY

General Section

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NEW CHIEF AT DENTAL AND ORAL PATHOLOGY DIVISION, ARMED FORCES INSTITUTE OF PATHOLOGY

CAPTAIN Louis S. Hansen (DC) USN, has been appointed Chief of the Dental and Oral Pathology Division at the Armed Forces Institute of Pathology, Washington, D. C. Captain Hansen, the first naval officer to head an Institute division, relieves Major General Joseph L. Bernier who was recently detached to assume the duties of Chief of the Army Dental Corps.

Captain Louis S. Hansen.

Captain Hansen received his B.S. and D.D.S. degrees from the University of Southern California and later completed an internship at the Los Angeles County Hospital. He has taken graduate work in oral pathology at the University of Michigan and at Georgetown University, where he received his M.S. degree. Captain Hansen is a diplomate of the American Board of Oral Pathology and a Fellow of the American Academy of Oral Pathology and the American Association for the Advancement of Science. In addition, he is a member of Omicron Kappa Upsilon honorary dental society and of Delta Sigma Delta fraternity.

Captain Hansen is a veteran of nineteen years' naval service. His last duty station was the U. S. Naval Dental School, National Naval Medical Center, Bethesda, Maryland, where he was engaged in dental research and served as an instructor in oral pathology.

Captain Hansen is married to the former Ignota Miller, a journalism graduate of the University of Southern California. They now reside with their three children in Bethesda, Maryland.

MYXOMA OF THE JAWS

Report of Two Cases

Nelson D. Large, D.D.S., Salisbury, N. C., and Harold H. Niebel, B.S., D.D.S.,** and Walter H. Fredricks, D.D.S.,*** Chicago, Ill.*

WHETHER or not a given type of tumor is of more than academic interest in oral diagnosis depends partly upon how often it will be seen in clinical practice. In the case of myxomas of the jaws, this is not often. Even so, reports of new cases do appear. A recent article by Zimmerman and Dahlin[6] covered twenty-six lesions of this type seen at the Mayo Clinic.

The earliest physical and x-ray changes of this form of myxoma might be detected during the course of a routine oral examination, while the lesion is subjectively asymptomatic. For this reason, its clinical behavior and some ideas as to its origin are of particular interest to the dentist.

In any form and in any location, the myxoma is rare. It is considered even more uncommon in the jawbones than in the rest of the skeleton. It is named for the mucouslike appearance of its stroma, which contains widely separated, immature, proliferating cells. There is no general agreement as to whether or not it represents a true neoplasm. In some instances an early diagnosis of myxoma is made and then changed to a final diagnosis of myxoid degeneration of some other lesion. It possibly represents a form of fibroma, but it demonstrates little or no mature collagen. Some of those that occur in the jaws are thought to arise from the embryonal type of connective tissue of the dental papilla, from undifferentiated mesenchymal stem cells of the bone itself, or from the periosteum. With these possibilities in mind, it is not surprising to find that some such tumors contain small spicules of calcified material which resemble either bone or bits of dental tissue.

When the calcified material present is identified as bone or as dentine or cementum, the lesion is classified accordingly as an osteogenic myxoma or an odontogenic myxoma.

A myxoma of the jaws usually lacks a well-defined border and is a locally aggressive, nonmetastasizing lesion. It has been observed that myxomas grow somewhat slower in the jaws than in other areas of the body. Here the prognosis is also enhanced by a better record of favorable response to wide local excision

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and curettement than is the case for treatment of myxomas generally. Radiation therapy is not effective for destruction of the process or for controlling its spread through the adjacent tissues. It is interesting to note that lesions occurring in the jaws apparently show no preference for any one sex or race, but the number of cases reported is too small for us to draw conclusions on either point.

As in the case of many lesions of the jawbones, the x-ray findings for myxomas are nonspecific. They are striking, however, and might resemble one of the various patterns for such lesions as central giant-cell tumors, fibrous dysplasias, or the ameloblastoma. In each of the two cases reported here, there were vague subjective symptoms which led to x-ray and tissue examination of the bones in question. In the first instance the tumor was in the maxilla. The tumor was located in the mandible of the second patient. In each case dental origin of the lesion was considered a possibility, even though the identity of the calcified material present was not clear-cut. Both lesions were recorded as osteogenic myxomas.

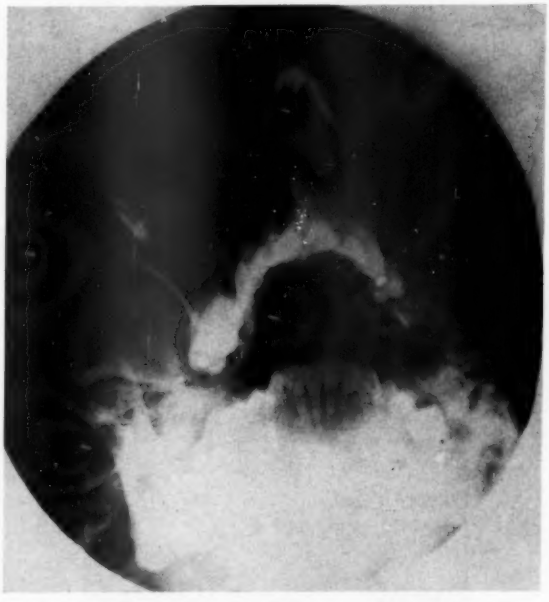

Fig. 1.—Obscuring radiopacity of left maxillary sinus.

CASE 1.—A 31-year-old man was admitted to the hospital following dental x-ray examination to determine the cause of pain in the left maxillary first molar region. The patient had noticed some enlargement of the left side of his palate for several months. Two months prior to his hospitalization he had been aware of left frontal headaches and a sensation of nasal stuffiness on the left side. He complained of a plugging sensation of the left ear and a nasal voice quality, all of which he attributed to and dismissed as sinus trouble.

Personal History.—The patient, a roofer by trade, smoked a package and a half of cigarettes a day and denied the habitual use of alcoholic beverages. He gave no history of past serious illness and had had no head and neck complaints prior to his present episode.

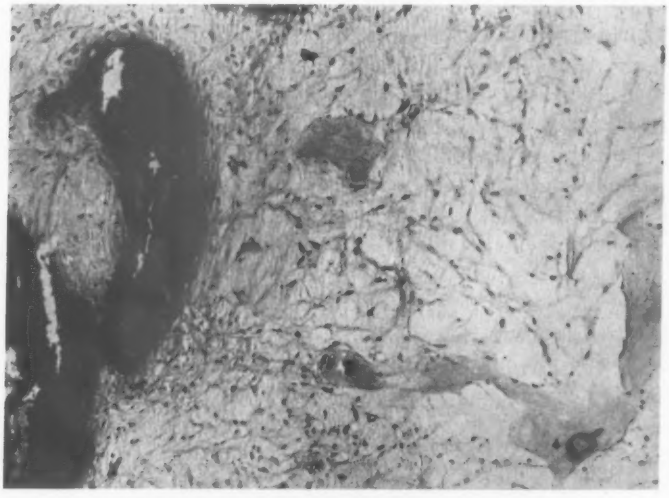

Fig. 2.—Myxoid stroma with osteoid formation.

Fig. 3.—Preoperative photograph.

Clinical Findings.—A firm 2 by 2.5 cm. swelling was noted on the left side of the hard palate, extending from the midline to the alveolar ridge. It was fixed and nontender. Teeth present in the left maxilla included the central and lateral incisors, the cuspid, and the first premolar. The premolar was slightly movable. The right and left cervical lymph nodes were palpable and indurated.

X-ray Findings.—A honeycomb type of radiolucent pattern was noted in periapical radiographs of the area involved. Views of the maxilla were characterized by an obscuring radiopacity in the area of the left maxillary sinus. The superior wall of this sinus appeared quite thin, and there was apparent erosion and partial destruction of its inferior lateral wall. The findings were interpreted as being suggestive of a destructive malignant neoplasm involving much of the left maxilla (Fig. 1).

Tissue Examination.—The initial microscopic examination consisted of the study of samples taken for frozen-section evaluation at the time of the operation for removal of the tumor.

A.

B.

Fig. 4.—*A,* Postoperative photograph of patient. *B,* Intraoral view of palate after operation.

The gross specimen was made up of a large portion of the left maxilla bearing a glistening gray tumor area. On cut section, the latter was found to resemble cartilage in consistency and again showed the glistening gray color of its surface. Several sections were obtained for further study.

Microscopic examination of these sections demonstrated a myxoid stroma with widely separated, relatively immature cells which were considered to be fibroblasts. The tissue had a noticeable embryonal quality and was punctuated throughout by foci of osteoid and new bone formation (Fig. 2).

Diagnosis: Osteogenic myxoma, maxilla.

Treatment and Course.—On the basis of the clinical data obtained and the results of frozen-section examinations during the operation, a resection of the maxilla was completed. The left zygoma was not disturbed. The patient's postoperative course was satisfactory, and he became free of pain in about five days (Figs. 3 and 4).

A dental obturator was designed to restore the missing teeth and correct the palatal defect. When this was being worn, the patient had good masticatory function and adequate correction of the speech difficulties resulting from the loss of a portion of the palate. There has been no evidence of a recurrence of the tumor after a follow-up period of four years (Fig. 5).

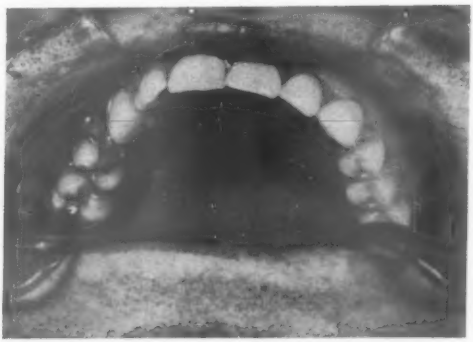

Fig. 5.—Intraoral view with obturator in position.

Fig. 6.—"Sunburst" pattern arising from periosteum, suggestive of a malignant bone lesion.

CASE 2.—A 35-year-old man was admitted to the hospital with complaints referable to a duodenal ulcer which had been diagnosed and treated an undetermined number of years in the past. Of more recent occurrence, but not of particular concern to the patient, was a slight paresthesia of the lower lip on the left side.

Personal History.—In addition to his account of recurrent episodes of abdominal pain over a period of many years, the patient stated that he had just recently noticed a slight numbness of his left lower lip. Four months prior to this he first became aware of some facial asymmetry. There was no history of trauma or previous treatment in this area.

Clinical Findings.—The patient had a duodenal ulcer which required treatment before attention could be focused on the oral findings. Oral examination was centered upon a

smooth, 4 by 6 cm. poorly demarcated, painless enlargement of the left mandible. It was fixed and extended from the lateral incisor to the molar region.

X-ray Findings.—Radiopaque lines with a vertical orientation to the mandible extended from the periosteum into the soft tissues. This "sunburst" effect was considered suggestive of a malignant bone lesion, possibly an osteogenic sarcoma (Fig. 6).

Tissue Examination.—Sections of a biopsy specimen consisted of a basophilic myxoid matrix containing immature stellate and spindle-shaped cells. The cells were well separated in this abundant stromal substance (Figs. 7 and 8). There were many spicules of osteoid material and occasional nondescript foci of calcification.

Fig. 7.

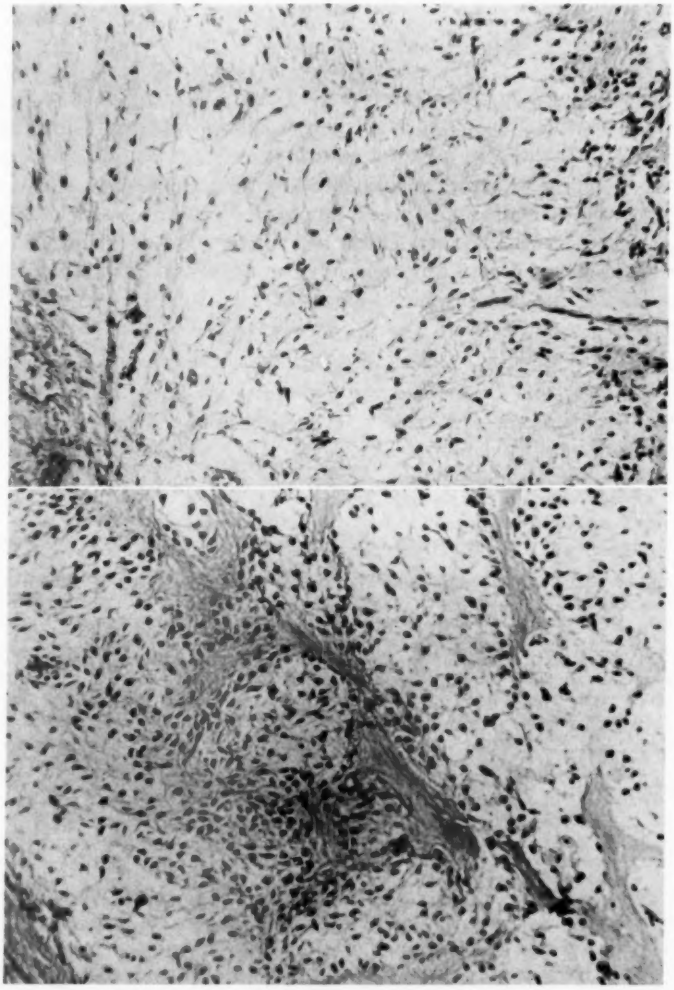

Fig. 8.

Figs. 7 and 8.—Myxoid matrix containing immature stellate and spindle-shaped cells.

Diagnosis: Osteogenic myxoma, mandible.

Treatment and Course.—The patient first received surgical treatment for his duodenal ulcer and was readmitted to the hospital about one month later for a mandibular resection. An attempt to restore the resected portion by an acrylic prosthesis was not successful. During subsequent hospital admissions plastic procedures were completed in preparation for an iliac bone graft. This plan was abandoned later, when the patient refused further proposals for a plastic repair.

Not quite four years following the mandibular resection, the patient returned for examination and treatment of a massive neoplasm of the sacrum. This was found to be a widespread chondrosarcoma with a somewhat myxoid stroma. It was considered to be a primary lesion, bearing no relation to the mandibular lesion. The latter showed no evidence of recurrence.

SUMMARY

1. An osteogenic myxoma was removed from the maxilla of a 31-year-old man, with apparent eradication of the lesion. Successful rehabilitation was accomplished with the help of a dental obturator.

2. An osteogenic myxoma was removed by partial resection of the mandible of a 35-year-old man. Four years later the patient developed a primary chondrosarcoma of the sacrum which led to his death. There had been no evidence of recurrence of the myxoma of the mandible. The two lesions were considered to be unrelated primary tumors.

3. Myxomas occurring in the jaws are thought to arise from cells of the dental papilla as well as from undifferentiated stem cells of the bone or periosteum.

4. The result of surgical removal of myxomas of the jawbones is somewhat better than is generally noted in the case of myxomas in other areas of the body.

REFERENCES

1. Bernier, J. L.: The Management of Oral Disease, St. Louis, 1955, The C. V. Mosby Company.
2. Dorland, W. A. N.: The American Illustrated Medical Dictionary, Philadelphia, 1951, W. B. Saunders Company.
3. Goldman, H. M.: Case Reports From the Files of the Registry of Dental and Oral Pathology, Am. J. Orthodontics & Oral Surg. 30: 265-314, 1944.
4. Stout, A. P.: Myxoma, the Tumor of Primitive Mesenchyme, Ann. Surg. 127: 706-719.
5. Weinmann, J. P., and Sicher, H.: Bone and Bones, ed. 2, St. Louis, 1955, The C. V. Mosby Company, pp. 401-403.
6. Zimmerman, D. C., and Dahlin, D. C.: Myxomatous Tumors of the Jaws, Oral Surg., Oral Med. & Oral Path. 11: 1069-1080, 1958.

COMPOUND COMMINUTED FRACTURE OF THE MANDIBLE

Report of a Case

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OPEN REDUCTION in the treatment of fractures of the mandible is extremely helpful in immobilizing badly displaced fragments. By this method more exact approximation of the fragments can be accomplished with less displacement by abnormal muscle pull. The risk of osteomyelitis has been greatly reduced since the advent of antibiotics.

The following case illustrates the use of intraoral wiring and splinting, direct wiring combined with open reduction, and an over-all stabilization by means of a Roger Anderson splint with its securing pins fixed in the mandible.

CASE REPORT

A white man, 24 years of age, was admitted unconscious to the West Lake Hospital on the evening of Jan. 10, 1954, approximately one hour after he had been in an automobile accident. His injuries were diagnosed as a possible basal skull fracture, a compound fracture of the mandible, and multiple facial lacerations.

History.—Police stated that a road block had been set up to halt a speeding car. In trying to escape, the driver had crashed into the road block. He was brought into the emergency room bleeding and unconscious.

Consultation Examination.—The patient was seen on January 16 for definitive care as requested by the neurosurgeon on the case after skull, scalp, and intracranial injuries had been ruled out. The patient had been unconscious for seventy-two hours.

Physical examination revealed a well-developed, well-nourished white man with several repaired facial lacerations on and below the chin and about the face and lips. There was subsiding edema, with periorbital ecchymosis and subconjunctival hemorrhage.

Oral examination revealed a segment of the right mandibular bone and alveolar process displaced into the floor of the mouth, obviously giving the patient a great deal of discomfort.

It was noted that the patient had been on a regimen of supportive and antibiotic therapy, consisting of intramuscular injections of 100,000 units of penicillin every three hours. He had been given a tetanus toxoid booster injection and placed on a controlled intake which consisted of a high-vitamin, high-calorie, liquid diet. The mouth had been kept as clean as possible.

Roentgenographic Examination.—The right lateral projection of the mandible revealed a severe comminuted fracture involving the entire half of the horizontal ramus of the mandible, extending from the molar region into the lateral incisor area. One part of the fracture

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could be seen to extend into the apex of the molar, and the anterior portion of the fracture extended into the region of the apex of the cuspid (Fig. 1). An occlusal anteroposterior roentgenogram revealed considerable medial displacement of the central fragment and much overriding of the distal fragment.

Laboratory Findings.—Blood studies on January 16 showed 5,200,000 erythrocytes and 10,600 leukoctyes, of which 72 per cent were polymorphonuclear neutrophils, 24 per cent were lymphocytes, and 4 per cent were monocytes. Hemoglobin was 16 grams. Bleeding time was three minutes, and clotting time was seven minutes. The Kahn test and the urinalysis were negative.

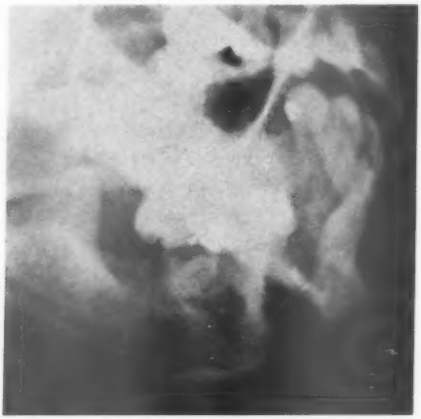

Fig. 1.—Lateral view of right mandible showing severe comminuted fracture involving entire half of horizontal ramus.

Treatment and Course.—As the patient was in distress, it was decided to adapt some maxillary and mandibular arch bars and secure them with 0.020 gauge stainless steel wire to relieve muscle tensions.

An attempt was made to reduce the parts by hand and immobilize them with elastic traction. The teeth were in good occlusion, but the right mandibular fragment was still displaced medially with much overriding of the remaining fragments.

It was decided that open reduction would accomplish the desired results. The patient was given pentobarbital sodium, 0.5 Gm., the night before surgery and nothing by mouth after midnight.

Operation.—On Jan. 17, 1954, after premedication with 1½ gr. pentobarbital sodium one hour before surgery and ½ gr. morphine sulfate with $\frac{1}{150}$ gr. atropine sulfate one-half hour before surgery, the patient was anesthetized with intravenous thiopental sodium and endotracheal nitrous oxide and oxygen. After the patient had been prepared and draped in the usual manner, an incision was made through the skin, subcutaneous tissue, and platysma muscle about one fingerbreadth below the right inferior border of the mandible, from a point just distal to the angle and curving slightly forward to a point below the mental foramen. The small vessels were clamped and tied off. Dissection was extended through the platysma muscle, exposing the external maxillary artery and vein. The latter were clamped and tied, as were the few remaining small vessels. The fracture segment was identified and dissected free. Drill holes were made, two in each segment, 1.5 cm. apart. Crisscross wires were used to approximate the segments, and a third wire was sewed to bundle the fragments together (Fig. 2).

Fig. 2.

Fig. 3.

Fig. 2.—Lateral roentgenogram of right mandible showing comminuted segments reduced and immobilized with transosseous wiring. A Roger Anderson external pin fixation is used to further reduce and stabilize the fragments.

Fig. 3.—Anteroposterior view of mandible showing reduction and immobilization employing internal transosseous wiring and external fixation with Roger Anderson pins secured in both distal and proximal fragments for added stability.

Because of muscle tension, it was decided to further stabilize and aid the immobilization of the mandible by means of a Roger Anderson splint with its securing pins fixed in both the distal and proximal fragments (Figs. 2 and 3).

The muscles were returned to their normal position with deep chromic sutures. The superficial structures were also closed with chromic catgut sutures, and the skin was carefully approximated with dermal sutures.

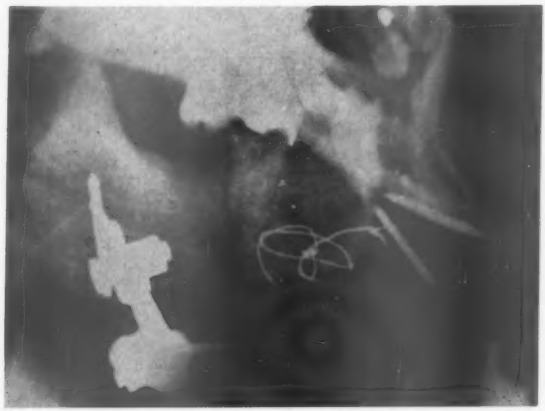

Fig. 4.—Postoperative lateral roentgenogram of mandible showing satisfactory alignment of fragments with slight evidence of callus formation. Maxillary and mandibular arch splints have been removed in anticipation of removing molar in line of fracture.

Fig. 5.—Lateral view of right mandible four months after operation, the external appliance having been previously removed. Note area of molar removal with good callus formation. There appears to be some separation of the major fragment, but callus formation is present.

Except for the usual postsurgical traumatic edema and ecchymosis, the patient had a normal postoperative period, aided by penicillin therapy. Cold compresses, bed rest, medication for pain, and adequate fluid intake were ordered. On the third postoperative day the patient's temperature was normal, and he was allowed partial ambulatory privileges. Every other extraoral suture was removed on the third postoperative day, and the rest were removed on the fifth postoperative day.

The patient was seen daily, and on Feb. 3, 1954, he was discharged. Postoperative roentgenograms revealed a severe comminuted fracture involving the entire half of the horizontal ramus of the right mandible reduced with four metallic pins and wires which were holding the fragments in good position (Fig. 4).

On March 4, 1954, the maxillary and mandibular arch bars were removed and the postoperative roentgenograms (Figs. 5 and 6) showed satisfactory alignment of the fragments with no definite evidence of callus formation. Clinical examination revealed a functioning mandible.

Fig. 6.—Anteroposterior view four months after operation revealing satisfactory progress.

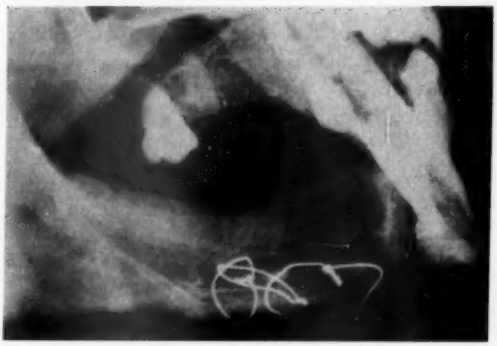

Fig. 7.—Lateral view of mandible some two years later, revealing a good functioning mandible.

The patient was followed periodically, and on March 18 the lower right first premolar and first molar were removed, as these were still potential sources of infection.

On March 23 the Roger Anderson pins were removed, and the patient was instructed to return for continued observation on May 25, 1954. On this date roentgenographic examination showed a marked improvement in that the portion of the fracture involving the molar

area on the right side had apparently healed completely. All of the previously described fixation had been removed, with the exception of the wire sutures about the portion of the fracture involving the cuspid area. There was still some separation of the major fragments in this area, but some callus could be seen.

Clinical examination some two years later showed a solidly healed mandible with good function. Roentgenographic examination verified this clinical impression (Figs. 7 and 8).

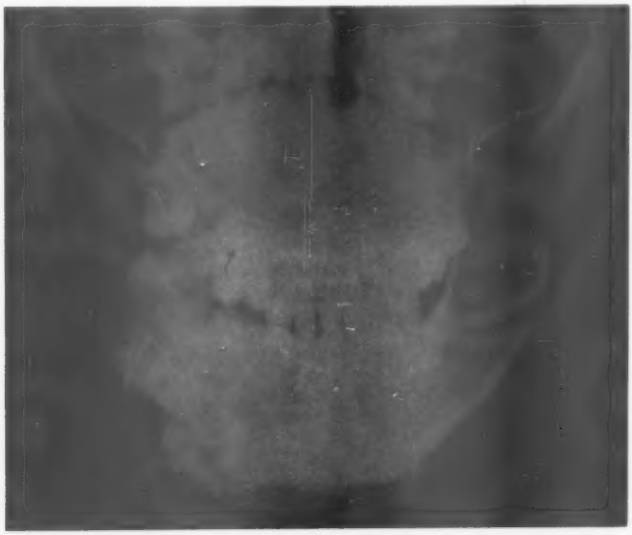

Fig. 8.—Anteroposterior view some two years after operation, showing a good union and a functioning mandible.

DISCUSSION

Open reduction of mandibular fractures is particularly useful in cases where there is a marked displacement. Transosseous wiring allows for a more accurate reduction and fixation, which would be difficult to manage by closed reduction. The Roger Anderson appliance is useful in giving additional support to the fragments, holding them in reduced position so that they cannot be displaced by muscle pull. The maxilla and the mandible should be immobilized by intraoral fixation.

CONCLUSION

A case of compound comminuted fracture of the right mandible which was treated by intermaxillary elastic traction and open reduction with transosseous wiring and additional fixation with a Roger Anderson appliance has been described.

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ENDODONTICS

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MANAGEMENT OF ROOT FRACTURE

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TRAUMATIC injuries involving the clinical crowns and the roots of permanent teeth of young persons present one of the greatest challenges in modern endodontics and restorative dentistry. All too often in these cases the main consideration is the loss of tooth substance or of the tooth itself, with failure on the part of the dentist to realize the possible psychological effects upon the patient.[1] These injuries are most frequently associated with accidents during play or participation in athletics and with bicycle or automobile accidents.

Many attempts at classification of fractured anterior teeth have been made in an effort to facilitate our understanding of the subject and to standardize treatment. Ellis[2] has contributed one of the most comprehensive classifications, including both the primary and the permanent dentitions.

The present report is limited to a discussion of *root fracture*. It is significant that root fracture seldom, if ever, occurs in young permanent teeth with wide-open apices. If the trauma were severe enough to fracture the root in these cases, luxation or complete avulsion would ordinarily result because of the as yet undeveloped periodontium and the small amount of root surface area. This report will therefore be concerned with root fracture in teeth that have completely formed roots and an alveolar housing.

The anatomic location of the fracture is an important consideration in prognosis. When fracture occurs in the coronal third of the root, the prognosis is poor because of the difficulty of immobilizing the fragments and the possible contamination of the fracture by bacteria through the gingival crevice. Fractures occurring in the middle third have a better prognosis if mobility is not a problem and if periodontal disease is not present. Fractures occurring in the apical third of the root have the most favorable prognosis when conservative treatment is administered. Actually, the term "conservative treatment" involves a gamut of procedures which may include stabilization of the fractured segments, root canal therapy, and the procedures necessary

for root resection, for all these methods are aimed at conservation of the teeth. The term "conservative treatment" as used in this article is defined as conservative to the extent that no root canal therapy or surgical intervention is instituted. Treatment is confined to stabilizing the teeth and allowing them to respond within the scope of physiologic wound healing. Root canal therapy is sometimes necessary when necrosis of the pulp is the sequela of root fracture; apicoectomy may or may not be indicated in these cases.

In order to understand how the pulp of a tooth with a fractured root can survive, it is necessary to review the biology and histology of the pulp, together with the pathologic changes brought about by trauma and the nature of the healing process. The pulp is the highly vascular formative organ of the tooth, and it consists of connective tissue, blood vessels, lymphatic vessels, nerves, and highly specialized peripheral odontoblasts. Histologically, the cellular components of the pulp are the odontoblasts, fibroblasts, histiocytes, wandering cells, and undifferentiated mesenchymal cells. These latter three types of cells act as macrophages when the pulp is in an inflammatory stage. The intracellular substance of the connective tissue consists of small collagenous fibers dispersed in a gelatinous ground substance. The capillary vessels are narrowed to the extent that red blood cells move through them one at a time in single file. The lymphatic system is not organized per se but consists of intercellular spaces into which lymph may drain. The nerve supply of the pulp enters through the apical foramen as tiny medullated fibers that are found along the blood vessels; some of these fibers also extend around the odontoblasts and seem to terminate in the tubules of the predentine.

Trauma of varying degrees may cause hyperemia, pulpitis, and subsequent necrosis of the pulp due to the intricate make-up of the blood vessels within the pulp and the biochemical mechanisms involving inflammation that are triggered by the irritation. Because of the irritation, there is an increase in the permeability of the capillary wall, which results in leaking of plasma. Hemorrhage from the small vessels results in their engorgement and hyperemia due to slowing of the circulation. Hyperemia of the pulp, if not excessive, is reversible; in other words, the pulp may recover completely if the irritant is removed. If, on the other hand, the irritant is excessive, the pulp will degenerate with eventual necrosis as the result of a thrombotic phenomenon.[3]

The nature of the inflammatory response is the same as that found in other tissues, irrespective of the irritant. In inflammation, the gross clinical manifestations or symptoms are reflections of the microscopic changes. Microscopically, the changes from hyperemia to pulpitis include the characteristics of an acute inflammation: (1) dilatation of the blood vessels, (2) increased permeability of the capillary wall, (3) slowing of the circulation due to the loss of viscosity of the blood (sludging of the red blood cells), (4) accumulation of leukocytes along the peripheral wall of the vessels (margination), and (5) migration of the white blood cells through the vessel walls (diapedesis).

With the congestion of the vessels and the slightest amount of accompanying edema, the pressure of the inflammatory response within the unyielding

dentinal walls is such that degeneration ensues. The severity of the inflammatory process in root fracture is reduced, since the space between the segments provides an avenue of escape for the increased blood supply. It has also been shown that when the root is fractured there is sometimes established a collateral communication between the circulation of the periodontal tissues and that of the pulp, which relieves the congestion of the small vessels and makes the prognosis more favorable.

The healing of traumatized tissue involves two processes—repair and regeneration. The repair process is actually a terminal phase of the inflammatory process and has to do with the control of hemorrhage within the pulp, the organization of the blood clot, and elimination of the damaged tissue. Immediately following the traumatic insult of root fracture, the space between the fragments is flooded with blood. This is then followed by the clotting mechanism. The elimination of the damaged tissue is brought about by action of the macrophages, which are the histiocytes, wandering cells, and mesenchymal cells of the pulp. In regeneration, the fibroblasts organize to bridge the fragments with fibrous tissue. Menkin[4] states that these fibroblasts grow in abundance, once the irritant has been removed by the macrophages. Their growth is accompanied by the proliferation of the capillary endothelium, for the fibroblasts need a vital blood supply. This phase of regeneration is often referred to as granulation tissue, a tissue that is quite resistant to superimposed infections. The formation of fibrous tissue appears to be an intermediate stage of healing, although many cases in which this tissue has been maintained for many years have been reported. In most cases, however, this fibrous tissue is replaced with a calcified substance (a cicatrix or scar tissue, which histologically resembles cementum and dentine) through the action of the odontoblasts and cementoblasts. The processes of repair and regeneration in root fracture closely resemble those seen in a typical bone fracture, all the clinical requirements having been met.

CASE REPORTS

CASE 1.—A 10-year-old girl had a bicycle accident and arrived at the dental office with the maxillary right central incisor partially avulsed. Roentgenographic examination revealed a fracture in the apical third of the root (Fig. 1, *A*). Treatment consisted of reducing the fracture (Fig. 1, *B*) and stabilizing the tooth by means of a plastic splint covering the incisal and occlusal surfaces of all the upper teeth. Prophylactic antibiotic therapy was administered in an effort to prevent possible bacterial invasion via the gingival sulcus or the pulpoperiodontal hematogenous route. The plastic splint was left in position for three weeks; it was then removed and replaced by a simple orthodontic wire splint which remained in place for another three weeks. The postoperative roentgenograms taken one year and two and one-half years later are shown in Fig. 1, *C* and *D*. When the patient was last seen, the tooth responded to vitality tests within the normal range, the color was unchanged, and there was no mobility (Fig. 1, *E*).

CASE 2.—A 14-year-old boy ran into an iron reinforcing bar on a swing at school. Some three months later he complained that his tooth was "loose when [he] whistled," and at this time he visited the dental office for treatment. Clinically, the tooth gave a positive response to percussion, ice, and heat, and the vitalometric response was well within normal limits. Roentgenographic examination revealed a fractured root of the maxillary

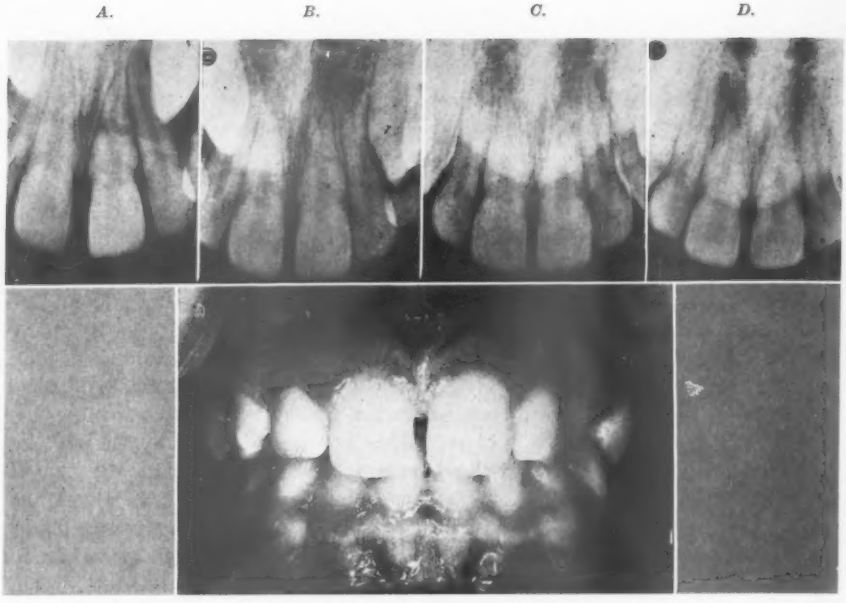


Fig. 1.—Case 1. *A*, Fractured root of the maxillary right central incisor showing partial avulsion of the coronal fragment (June 11, 1957).
B, Reduction of the fracture previous to splinting (June 11, 1957).
C, Postoperative result approximately one year later (May 17, 1958).
D, Postoperative result on Jan. 4, 1960. Note regeneration of "cellular dentine."
E, The maxillary right central incisor as seen clinically on Jan. 4, 1960.

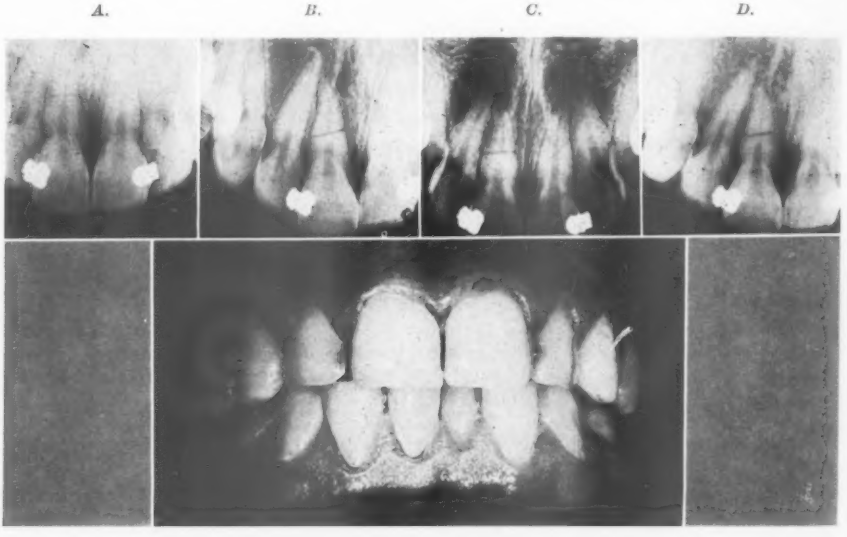


Fig. 2.—Case 2. *A*, Roentgenogram revealing fracture of maxillary left central incisor (June 21, 1956). Note tendency to repair.
B, One year after the accident.
C, Two years after the accident.
D, Roentgenogram showing regeneration of "cellular dentine" (Jan. 6, 1960).
E, Clinical appearance showing the normal color of the maxillary left central incisor almost four years after the accident.

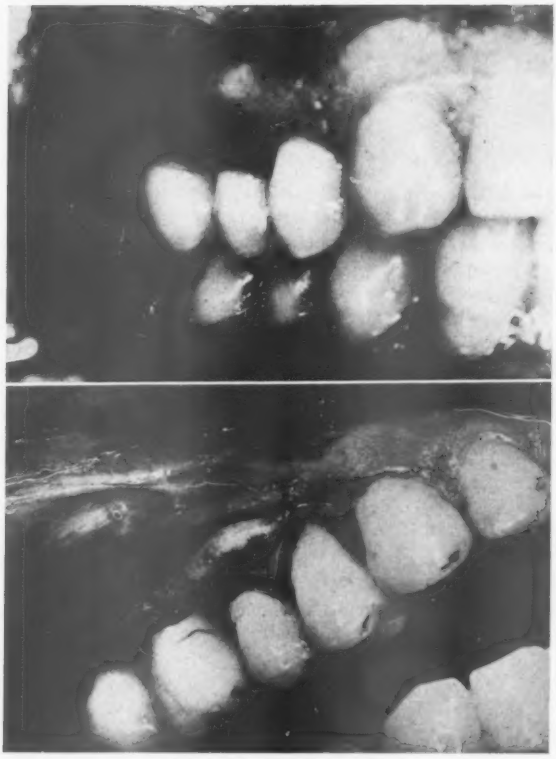
left central incisor with some evidence of repair (Fig. 2, *A*). Treatment consisted of reshaping the tooth to eliminate undue occlusal stress in centric, protrusive, and lateral excursions. Since there was no mobility and there was roentgenographic evidence of repair, splinting was not necessary. Four years after the accident the tooth was normal in color, it responded to the vitalometer test the same as the adjacent central incisor, and there was no mobility (Fig. 2, *D* and *E*).

CASE 3.—A 30-year-old man was in a serious automobile accident on Dec. 18, 1955. Among other injuries, he sustained multiple fractures of the right maxilla, including the facial and orbital surfaces and also the zygomatic process. Reduction of the fractures and the necessary cosmetic corrections required six maxillofacial operations, all of which covered a span of sixteen months. During this time, there was no dental treatment of any kind. On April 25, 1957, the patient came to the dental office for treatment, and a full-mouth roentgenographic survey was made. Clinically, it was observed that some of the buccal cusps of the maxillary and mandibular molars had been sheared away by the impact of the accident. With the aid of a periodontal probe, it was found that the periodontal problem was of primary importance. The pronounced exostosis on the buccal aspect of the maxilla, together with food impaction over an extended period of time, contributed to considerable pocket depth in the premolar and molar areas, ranging from 5 to 7 mm. The patient was then referred to a periodontist for correction of the periodontal defects. Correction in this case included reshaping the bone to establish a more physiologic contour, making it possible to eliminate the pockets. When the initial incision was made preparatory to osseous surgery, a longitudinal fracture of the buccal root of the maxillary first premolar could be seen (Fig. 3, *E*). This fracture had not been diagnosed roentgenographically, partly because the tooth had been asymptomatic for approximately two years (Fig. 3, *D*). The periodontal surgery was completed, and it was decided that the premolar involved with the fractured root should be observed. Vitalometric tests on the buccal and lingual surfaces still elicited a positive response. Because of the inaccuracy of the vitalometer in testing multirooted teeth, a bur test without anesthesia was instituted; this also gave a positive response (Fig. 3, *B* and *C*). Four years after the accident, the tooth showed no sign of rarefaction at the apex roentgenographically, it was normal in color, and it responded to vitality tests within the normal range as compared with the corresponding mate in the arch as well as with the adjacent teeth.

CASE 4.—A 9-year-old boy had a bicycle accident in May, 1956. Clinical examination revealed a deep dentinal fracture of the maxillary left central incisor; the roentgenogram appeared to be negative. At the time of the emergency visit the patient received palliative treatment only. On Oct. 27, 1956, there was an acute exacerbation of the involved tooth; drainage was established through the tooth by means of a lingual opening. On Sept. 4, 1957, the patient was referred for endodontic treatment. Roentgenograms revealed a periapical area and two smaller lateral areas of roentgenolucency (Fig. 4, *A*), which seemed to indicate a fractured root although the fracture line was not well defined. Tangential roentgenograms substantiated the presence of the areas, but the line of fracture was still not clearly defined. Because of the age of the patient and the strategic importance of the tooth, obturation of the canal was completed on Sept. 18, 1957, by the lateral condensation technique. Two negative bacteriologic cultures were obtained before obturation. Fig. 4, *B* shows the obturation completed, with the sealer forced between the fractured segments. Postoperative roentgenograms one year later and two and one-half years later can be seen in Fig. 4, *C* and *D*. Because of the anatomic location of the fracture in this case, root canal therapy must be classified as a holding measure only. The prognosis for this tooth is not at all favorable, but the treatment in this case was expedient. Mobility in this case is minimal; hence, the boy can be expected to retain the tooth until a fixed prosthesis is contemplated.

A. *B.* *C.*

D.



E.

Fig. 3.—Case 3. *A,* Presurgical roentgenogram illustrating the difficulty of diagnosing the longitudinal fracture of the root of the maxillary right first premolar (Aug. 10, 1957).

B, Postsurgical roentgenogram showing the fractured root more clearly.

C, Note absence of periapical area and the bur test cavity used to establish vitality (Oct. 7, 1959).

D, Preoperative photograph showing "ledging" of the maxillary bone.

E, Initial incision preparatory to osseous reshaping of bone to correct periodontal defects. Note longitudinal fracture of the buccal root of the maxillary first premolar. (Courtesy Dr. John F. Prichard.)

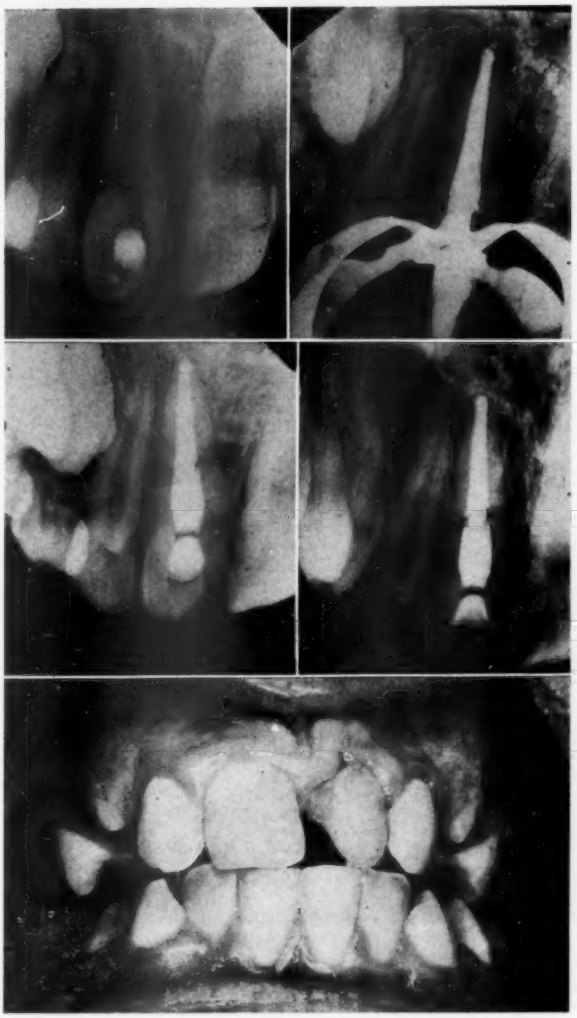


Fig. 4.—Case 4. *A*, Preoperative roentgenogram of the maxillary left central incisor (Sept. 4, 195). Note periapical roentgenolucent area and two lateral areas.

B, Obturation of the canal (Oct. 15, 1957). Note the sealer forced between the fractured segments and the size of the lateral roentgenolucent areas.

C, Postoperative roentgenogram one year later showing lateral areas somewhat decreased in size.

D, Fractured segments showing slight separation, lateral areas greatly reduced (Aug. 31, 1959). Roentgenogram, as compared with that in *A*, shows more coronal tooth structure fractured away as a result of a subsequent accident.

E, Photograph showing the clinical appearance of the tooth. Note the eruption of the two cuspids as compared with Fig. 4, *A*. (Courtesy Dr. W. H. Rickles.)

CASE 5.—A young man, 18 years of age, was in an automobile accident and was thrown against the steering wheel. The full impact hit him in the mouth. The patient was referred to the office for endodontic treatment of the maxillary left central incisor about six months following the accident. Examination disclosed a fracture of the root of the left lateral incisor. This case is reported to emphasize some of the important diagnostic signs in root fracture and their influence on prognosis, which in this case was unfavorable. The lateral incisor responded to the vitalometric test, ice, and heat the same as the corresponding tooth in the arch, although roentgenographically there appeared to be some widening

Fig. 5.—Case 5. *A*, Roentgenogram showing large periapical lesion of the maxillary left central incisor and the fractured root of the maxillary left lateral incisor (June 10, 1959).

B, Postoperative roentgenogram showing obturation of the root canal of the tooth with a deep dentinal fracture (July 10, 1959). Note widening of the periodontal membrane space of the lateral incisor.

C, Clinical appearance showing the position of the lateral incisor in the arch. Note that the teeth are in centric occlusal position.

of the periodontal membrane space. It is significant to note that the adjacent central incisor, with a coronal fracture into the dentine, showed a periapical area of bone absorption and did not respond to vitality tests, whereas the tooth with the fractured root was vital. This illustrates the fact that teeth with fractured roots often have a better chance of survival than those with crowns that are fractured well into the dentine. One reason for this is that the protoplasmic extensions of the odontoblasts are exposed in deep dentinal fractures, which in essence is a pulpal exposure, and therefore these cases should be treated as such. The lines of fracture in this case were located at the juncture of the

middle and coronal thirds, resulting in unfavorable alveolar support for the coronal portion. The approximation of the fragments was poor, and it is conceivable that there could have been some contamination at the lateral borders of the fracture from the gingival crevice. Of further importance in treatment planning was the fact that, because of its position in the arch, the tooth did not lend itself to good physiologic form from a periodontal standpoint and, esthetically, there was much to be desired. Because the basic requirements for successful therapy were not fulfilled and the tooth was not of strategic importance, it was sacrificed and a bridge was constructed.

DISCUSSION

Many investigators have contributed to our knowledge of root fracture,[5-8] so that a favorable prognosis may be predicted in most cases. According to Kronfeld,[6] there are four basic clinical requirements for successful therapy:

1. The fragments must be in apposition.
2. The fragments must be immobilized in this position by splinting.
3. There must be an absence of infection.
4. The patient's general health must be such as to make reparative and regenerative processes possible.

Blackwood,[9] in a recent study illustrated with photomicrographs, has explained the reparative and regenerative processes in root fracture and identified the calcified tissue as "cellular dentine." This kind of tissue can be seen in the roentgenograms of the cases presented (Figs. 1, *D* and 2, *D*).

Dietz,[10] has referred to these fractures as "sarcophagizing" the pulps; hence, observation at yearly intervals, with vitalometric tests and roentgenographic examination, is indicated. The patient's parents should be instructed to watch for incipient discoloration, which indicates that "sarcophagization" has occurred. This phenomenon is ideal from an endodontic point of view. If discoloration has occurred, endodontic therapy, followed by a bleaching procedure, may be necessary.

SUMMARY

1. An effort has been made to establish a rationale for the conservative approach in the treatment of root fracture in permanent teeth.

2. The requirements for success in the treatment of root fracture are enumerated.

3. The case histories presented substantiate the findings of many that the prognosis is often favorable in cases of root fracture.

REFERENCES

1. Kaplan, Alex H.: Psychological Factors in the Practice of Dentistry, J. Am. Dent. A. 57: 835, 1958.
2. Ellis, Roy G.: The Classification and Treatment of Injuries to the Teeth of Children, ed. 3, Chicago, 1957, Year Book Publishers, Inc., p. 169.
3. Seltzer, S., and Bender, I. B.: Inflammation in the Odontoblastic Layer of the Dental Pulp, J. Am. Dent. A. 59: 720-725, 1959.
4. Menkin, V.: Biochemical Mechanisms in Inflammation, ed. 2, Springfield, Ill., 1956, Charles C Thomas Publishers, p. 295.

5. Kronfeld, R.: A Case of Tooth Fracture With Special Emphasis on Tissue Repair and Adaptation Following Traumatic Injury, J. D. Res. 15: 429, 1935-36.
6. Boyle, Paul E.: Kronfeld's Histopathology of the Teeth and Their Surrounding Structures, ed. 4, Philadelphia, 1955, Lea & Febiger, p. 445.
7. Sommer, R. F., Ostrander, F. D., and Crowley, M. C.: Clinical Endodontics, Philadelphia, 1956, W. B. Saunders Company, pp. 237-373.
8. Rabinowitch, B. Z.: The Fractured Incisor, Pediat. Clin. North America 3: 979-994, 1956.
9. Blackwood, H. J. J.: Tissue Repair in Intra-Alveolar Root Fracture, ORAL SURG., ORAL MED. & ORAL PATH. 12: 360-370, 1959.
10. Dietz, Victor H.: Endodontic Surgery, North-West Dent. 37: 207-209, 1958.
11. Prichard, John F.: Personal Communications.

303 SOUTH WASHINGTON ST.

ROOT FRACTURE*

Report of a Case

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INTRODUCTION

SUCCESSFUL endodontic treatment is dependent upon a complete diagnosis. The process of arriving at this diagnosis should include a thorough evaluation of the case history and clinical examination, as well as an evaluation of the underlying bone structure as shown by a properly exposed roentgenogram.

Too often the use of roentgenograms is taken for granted. They are thought to be infallible, and thus they are often used as the sole means of arriving at a diagnosis and treatment plan.

The following case illustrates some problems that arise in correlating roentgenograms with other findings. It also shows the difficulty of arriving at the correct diagnosis and hence the correct treatment plan.

CASE REPORT

P. L., a 9-year-old boy, came to the Therapeutic Clinic, University of Illinois College of Dentistry, on Oct. 7, 1957, complaining of a sore and loose tooth and the presence of a "gumboil." He reported that the tooth was particularly sore when he was eating or when something cold was applied and that his gingivae bled in the morning when he woke up.

The patient's complaint resulted from a blow to the face that he received late in August, 1957. Two days after the accident his badly swollen face and lip were examined by a physician, and the boy was treated with a single penicillin injection. The examination made at that time did not include roentgenograms. Three weeks later the "gumboil" developed. In spite of this, further treatment was not sought and the patient's condition remained relatively unchanged up to the time that he came to the clinic.

On examination, the crown of the maxillary right central incisor showed the loss of one-fourth of the labial enamel plate, the loss of some underlying dentine, and a small exposure of the distal pulp horn. Gross facial swelling was not evident, but there was a small (2 by 2 mm.), elevated, fistulous opening on the attached gingiva, approximately at midroot, over the involved tooth. The anterior marginal gingivae showed a chronic inflammation and bled slightly when finger pressure was applied.

The tooth could be readily moved 1 to 2 mm. labially and lingually, but this movement was extremely painful and accurate measurement of the mobility was not possible.

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When the fistula was probed, a small amount of pus was expressed and a bony cavity was found on each side of the tooth. Neither of the cavities extended to the apex. The tooth did not respond to the electric pulp tester, to hot compound, or to ethyl chloride spray.

These findings were thought to be compatible with the diagnosis of a chronic lateral periodontal abscess that had resulted from a necrotic pulp with a lateral canal or a root fracture.

However, the roentgenogram (Fig. 1) showed a maxillary central incisor of normal length, with a closed apex and a coronal roentgenolucency that was superimposed on a large pulp chamber. In the midroot area, mesially and distally, there was an area of rarefaction and loss of lamina dura. The bone around the apical third of the root was reasonably normal and intact. The periodontal membrane there was of normal thickness, although the lamina dura had lost some definition. Because of these findings and because neither a lateral canal nor a root fracture was seen, conservative endodontic therapy was elected.

Fig. 1.

Fig. 2.

Fig. 1.—Roentgenogram taken on Oct. 7, 1957. Note alveolar bone loss, mesially and distally, in midroot area and remaining alveolar crest bone. Labial enamel plate loss is superimposed on large coronal pulp chamber. The alveolar bone investing the apical third of the root is relatively intact and unchanged, as is the periodontal membrane.

Fig. 2.—Roentgenogram taken on Oct. 18, 1957. Note the reamer in the canal, which was used to aid root-length measurement. Note also the transverse midroot fracture and the reduced size of lateral roentgenolucencies. The reamer was placed in the canal through the labial fracture.

Treatment consisted of opening the tooth for drainage through the labial fracture and then irrigating the root canal and fistula. The root canal was left open for drainage. The irrigations were repeated four days later, and an antibiotic point was then sealed in the canal. The tooth seemed less mobile at this second appointment. One week later (October 18) the treatment was continued, and a reamer was placed in the canal to aid in measuring the root length. A roentgenogram taken with the instrument in place clearly showed a transverse midroot fracture (Fig. 2). With this finding, treatment was discontinued and the tooth was extracted in two pieces.

DISCUSSION

The roentgenogram used in arriving at the original treatment plan for this case was obviously inadequate. Although the alveolar bone loss was well

marked, the root fracture was not. The fracture could not be seen clearly even when the roentgenogram was rechecked later. Additional roentgenograms exposed from various angles or more pressure on the crown to open the fracture during filming might have localized the fracture. It seems likely that increased mobility of the fractured coronal portion caused some luxation which made the fracture line obvious in the roentgenogram taken one week later.

Generally, inflammation should have separated the segments and resolution allowed them to come together again. The opposite was true in this case.

A complicated and unsuccessful case involving endodontic therapy and a transverse root fracture has been presented. The fracture was not evident on the original roentgenogram, but it was seen on a subsequent one. The original diagnosis was incomplete as well as misleading and illustrates the fallibility of a single roentgenogram.

Section by the

ARMED FORCES AND PUBLIC HEALTH SERVICE

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PAROTID FLUID FLOW RATE

Its Relationship to pH and Chemical Composition

Ira L. Shannon, Major, USAF (DC), and John R. Prigmore, Captain, USAF (MSC)

INTRODUCTION

One of the most intriguing problems in glandular physiology is that concerned with the understanding of the excretory and/or secretory functions of the salivary glands. The mechanisms by which certain chemical constituents enter and are released by the gland cells are not at all clear. There is distinct disagreement as to whether or not there is active reabsorption by the duct cells and, conversely, whether or not certain duct cells actually elaborate chemical substances into the saliva. For over eighty years it has been known that the rate of flow from the gland exerts an influence on the concentrations of some of the chemical components of saliva, and many investigations have been carried out in flow rate-concentration relationships in efforts to clarify the mechanisms of glandular function. Unfortunately, there has been a marked diversity in duration and methods of glandular stimulation, procedures for chemical determinations, time of day of collections, dietary control, age of subjects, and other variables under the control of the investigator.

It was our purpose in the present study to investigate, under controlled experimental conditions, the effects of varying parotid gland flow rates upon the levels of bicarbonate, calcium, pH, inorganic phosphate, potassium, sodium, total protein, and chloride in parotid fluid. The performances of three different methods for chloride determination were also evaluated. Of further concern was the determination of intercorrelations for findings other than flow rate and

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The opinions expressed herein are those of the authors and are not to be construed as expressing official United States Air Force policy.

the development of a pattern of stimulation that would consistently provide a reproducible, well-spread flow rate pattern for this and future correlation studies.

PROCEDURE

This report includes the data from two separate flow rate experiments, each utilizing identical stimulatory procedures to elicit parotid flow. All subjects were young men between the ages of 17 and 22 years; each had recently undergone physical examination and had been found fit for military service. Dietary factors, hours of waking and sleeping, physical exertion, and environmental exposure were identical within the experimental groups. Admitted recent bouts of upper respiratory infection, nausea, vomiting, diarrhea, constipation, or any significant systemic symptom disqualified a prospective subject.

Ten-minute parotid fluid test samples were collected by vacuum cap from the right parotid gland with the following sequence of stimulants: Sample 1, one rubber band (size 32); Sample 2, one-half stick of gum base*; Sample 3, one and one-third sticks of sugared gum*; Sample 4, one-half stick of gum base; Sample 5, one rubber band (size 32). This procedure represented an attempt to study flow rate-concentration relationships with significantly different rates of flow in both ascending and descending order. All samples were collected under mineral oil with the collection tube packed in ice. Prior to collection of each test specimen, a ten-minute accommodation (waste) sample was collected with the identical eliciting agent and discarded. The sugared gum bolus was renewed at the expiration of five minutes. The different quantities of gum base and sugared gum were employed since the hydration of one-half stick of gum base over a five-minute period produces a final bolus volume approximating that found for one and one-third sticks of sugared gum, the decrease in volume of the latter being due primarily to the rapid loss of sugar from the gum bolus.

FLOW RATE EXPERIMENT 1

In the first experiment five test samples were collected from each of twenty-eight subjects, and levels were determined for rate of flow, bicarbonate, calcium, pH, and inorganic phosphate. In the next phase twenty-five subjects were sampled and observations were recorded for flow rate, potassium, sodium, total protein, and chloride. Volume was read directly from the graduated centrifuge tubes in which collections were made; pH was determined on a Beckman Zeromatic pH meter; bicarbonate was measured by the method of Van Slyke[1]; calcium and inorganic phosphate were determined on the Autoanalyzer† by methods that we have previously reported.[2] Sodium and potassium analyses were performed on a Beckman DU flame spectrophotometer as previously described.[3] Total protein levels were ascertained by the biuret[4] reaction. Chloride concentrations were determined on the Cotlove[5] chloridometer,‡ and an evalua-

*Furnished by Dr. Robert Heggie, American Chicle Co.
†Technicon Instruments Corp., Chauncey, New York.
‡Laboratory Glass and Instrument Corp., New York, New York.

tion of this method as compared to the Autoanalyzer method and an iodometric titration method was included. Since several of our previous reports[6-12] and those of other investigators[13, 14] have been based upon the Van Slyke and Hiller[15] modification of the method of Sendroy[16] for chloride determination, and since we were aware[12] of a discrepancy between chloride findings and actual concentration by this method at low levels, the Sendroy procedure was the hand method chosen for comparison.

In the statistical analysis of the flow rate-concentration data, correlation coefficients were computed for each pair of variables for each subject, based upon the five pairs of observations. Then an average "within-subjects" correlation coefficient was calculated for each pair of variables. In addition, means and standard deviations of the five observations were computed for each variable and for each subject, and a "between-subjects" correlation coefficient was calculated for each pair of variables. The probability of significance was determined for each coefficient.

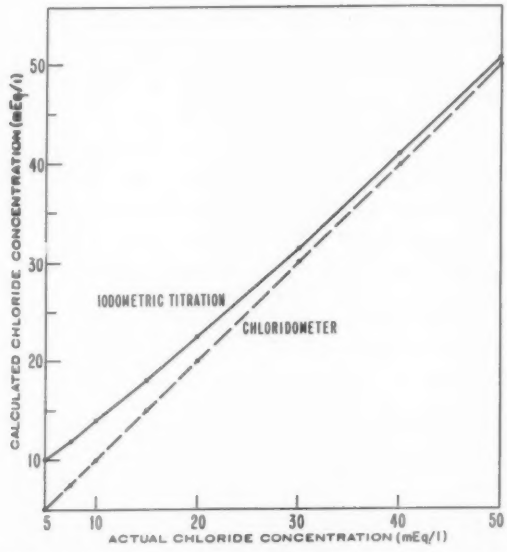

Fig. 1.—The chloridometer versus Sendroy titration in chloride determination.

Results and Discussion.—

Chloride methods: In evaluating the three methods for the determination of parotid fluid chloride concentration, the Autoanalyzer portion was discontinued for two reasons: (1) A drift in the recorder response to repeated running of standards made accurate estimation difficult and (2) when the untreated saliva was mixed with the sulfuric acid-hyamine diluent, a turbidity was produced which led to the plating-out of a white precipitate upon the glass wall

of the cactus-tree fitting in which the mixing took place. A comparison of the iodometric titration chloride method to the Chloridometer method is shown in Fig. 1, a graph which plots actual chloride concentrations of 5 to 50 mEq. per liter chloride standards against chloride concentration for these standards as calculated by titration and by the machine. Titration values were found to be higher, this effect being especially pronounced at the lower levels of concentration where a standard of actually 5.0 mEq. per liter chloride concentration gave a titration value of 10.1 mEq. per liter. Between 5.0 and 15.0 mEq. per liter the titration results were linear, while between 15.0 and 50.0 mEq. per liter the results, although linear, were on a slightly different plane. From 60.0 to 100.0 mEq. per liter neither method varied more than 0.5 per cent from the actual standard value and no systematic pattern of difference was found. Throughout the range of from 5.0 to 100.0 mEq. per liter the Chloridometer values were virtually identical to the actual chloride concentration. The difference in performance of the two methods on a series of parotid fluid samples was very similar to that found with standards.

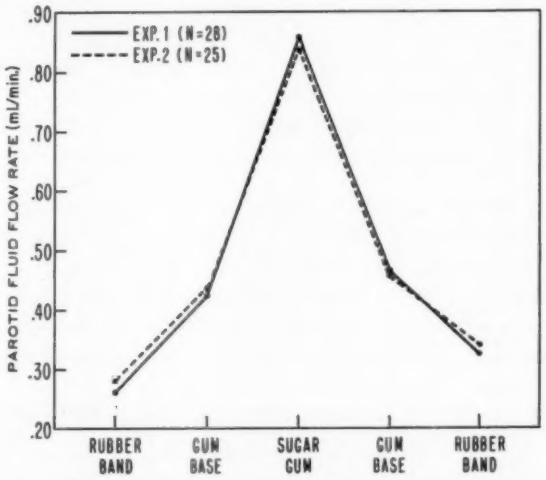

Fig. 2.—Reproducibility of the elicitation procedure.

Performance of stimulation procedure: Flow rate, as it was produced in these studies, was spread sufficiently to provide an adequate range for correlation studies. That the reproducibility of this method is of a high order is demonstrated when the results for the two experiments are compared (Fig. 2). In the first study a rubber band flow rate mean of 0.261 ml. per minute (S.D., 0.107) increased to 0.425 (S.D., 0.139) with gum base and to 0.858 (S.D., 0.227) with sugared gum and fell to 0.465 (S.D., 0.165) and to 0.326 (S.D., 0.144) when gum base and rubber bands were repeated. Comparable figures for the second experiment were 0.279 ml. per minute (S.D., 0.121), 0.436 (S.D., 0.194), 0.838 (S.D., 0.284), 0.458 (S.D., 0.191), and 0.340 (S.D., 0.147), respectively.

On the basis of these findings, this elicitation procedure could be recommended as satisfactory with respect to simplicity, time required, flow rate range, and reproducibility. It is extremely difficult to interpret flow rate correlation studies with stimulation patterns providing fewer degrees of freedom for statistical analysis.

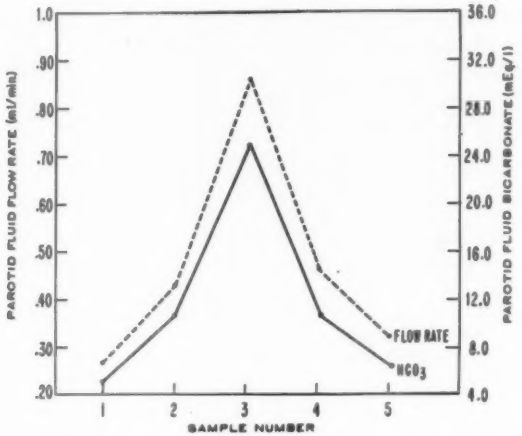

Fig. 3.—Correlation between flow rate and bicarbonate.

Bicarbonate: Within subjects a significant ($P < .01$) positive correlation ($r = 0.98$) was found between flow rate and bicarbonate (Fig. 3). For the five samples the bicarbonate mean values were 5.06 (S.D., 4.49), 10.65 (S.D., 5.94), 24.77 (S.D., 7.54), 10.52 (S.D., 5.30), and 6.23 (S.D., 4.38) mEq. per liter. Bicarbonate was found to be positively correlated with calcium and with pH and to be negatively correlated with inorganic phosphate (Table I). When

TABLE I. THE AVERAGE "WITHIN-SUBJECTS" CORRELATION COEFFICIENTS FOR FIVE PAROTID FLUID SAMPLES FROM EACH OF TWENTY-EIGHT MALE SUBJECTS AND THE PROBABILITY OF COEFFICIENT SIGNIFICANCE

	BICARBONATE	CALCIUM	pH	INORGANIC PHOSPHATE
Flow rate	0.98 ($< .01$)	0.55 ($< .01$)*	0.93 ($< .01$)	−0.86 ($< .01$)
Bicarbonate		0.54 ($< .01$)*	0.93 ($< .01$)	−0.86 ($< .01$)
Calcium			0.27 ($< .05$)	−0.29 ($< .05$)*
pH				−0.87 ($< .01$)

*The correlation between these two variables differed significantly from one subject to another.

an entry in Table I was marked with an asterisk, the correlation between these two variables had been found to differ significantly from one subject to another, and therefore the average was not an estimate of the correlation for a given person. The average correlations not marked with an asterisk were estimates of the correlation coefficients for all subjects in the population. In other words,

from the data in these experiments, there was no reason to suspect that the correlations not marked with an asterisk differed from one subject to another. In the "between-subjects" correlations bicarbonate and flow rate demonstrated a significant positive correlation ($r = 0.59$), but the correlations of bicarbonate with calcium, pH, and phosphate were not significant (Table II).

TABLE II. THE "BETWEEN-SUBJECTS" CORRELATION COEFFICIENTS AND THE PROBABILITY OF THEIR SIGNIFICANCE

	BICARBONATE	CALCIUM	pH	INORGANIC PHOSPHATE
Flow rate	0.59 ($< .01$)	−0.36 ($> .05$)	0.45 ($< .05$)	−0.06 ($> .05$)
Bicarbonate		−0.28 ($> .05$)	0.32 ($> .05$)	0.23 ($> .05$)
Calcium			−0.60 ($< .01$)	−0.04 ($> .05$)
pH				0.29 ($> .05$)

Calcium: The positive correlation coefficient for flow rate and calcium ($r = 0.55$), calculated within subjects, was significant at the 1 per cent level, but it differed significantly from one subject to another (Fig. 4). The mean calcium values for the five specimens were 1.61 (S.D., 0.47), 1.45 (S.D., 0.36), 1.70 (S.D., 0.36), 1.39 (S.D., 0.30), and 1.50 (S.D., 0.47) mEq. per liter. Calcium was positively correlated (Table I), with bicarbonate ($P < .01$) and pH

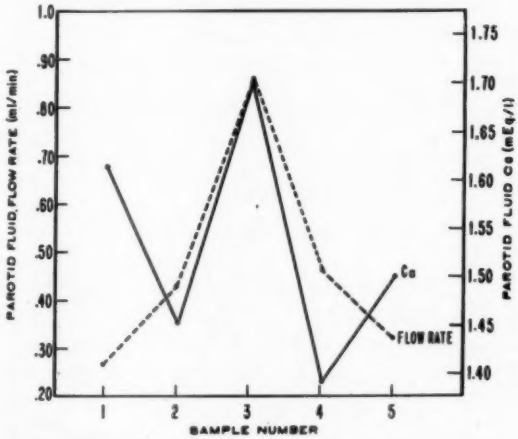

Fig. 4.—The correlation between flow rate and calcium.

($P < .05$) and negatively correlated with phosphate ($P < .05$). In the "between-subjects" correlations, calcium was negatively correlated with flow rate ($P < .01$) and negatively correlated with pH ($P < .01$), while the correlations with bicarbonate and phosphate were not significant (Table II).

pH: The positive correlation, within subjects, between pH and flow rate was significant at the 1 per cent level (Fig. 5). Means for pH for the twenty-eight subjects were 6.77 (S.D., 0.37), 7.07 (S.D., 0.27), 7.38 (S.D., 0.17), 7.08 (S.D.,

0.22), and 6.88 (S.D., 0.29). pH was found to be positively correlated with bicarbonate ($P < .01$), positively correlated with calcium ($P < .05$), and negatively correlated with phosphate ($P < .01$) (Table I). In the "between-subjects" analysis, pH was positively correlated with rate of flow ($P < .01$) and negatively correlated with calcium ($P < .01$). The correlations with bicarbonate and with phosphate were not significant (Table II).

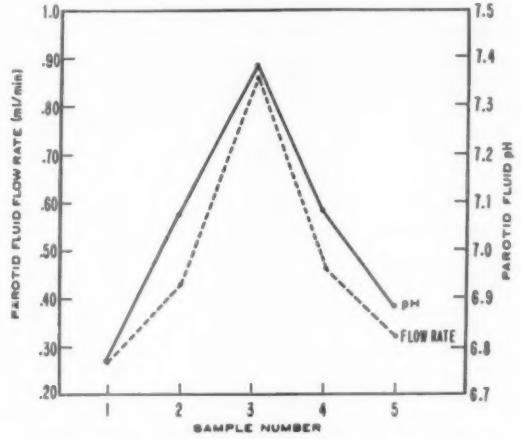

Fig. 5.—The correlation between flow rate and pH.

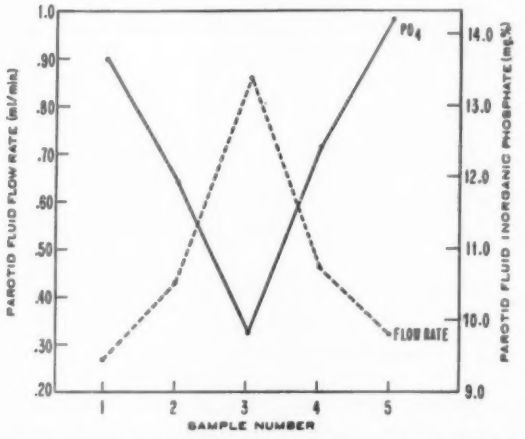

Fig. 6.—The correlation between flow rate and inorganic phosphate.

Inorganic phosphate: A strong negative correlation ($P < .01$) was noted within subjects between phosphate and flow rate (Fig. 6). Means for phosphate were 13.58 (S.D., 2.40), 11.88 (S.D., 2.03), 9.77 (S.D., 1.96), 12.36 (S.D., 2.40), and 14.20 (S.D., 265) mg. per 100 ml. of parotid fluid.

Phosphate was negatively correlated with bicarbonate ($P < .01$), calcium ($P < .05$), and pH ($P < .01$) (Table I). "Between-subjects" phosphate (Table II) was not significantly correlated with any of the four other variables under study.

FLOW RATE EXPERIMENT 2

Potassium: In the "within-subjects" analysis no significant correlation could be found for potassium with flow rate (Fig. 7), sodium, or chloride. A positive correlation of borderline significance ($P < .05$) was noted for potassium and total protein (Table III). For the twenty-five subjects, the potassium

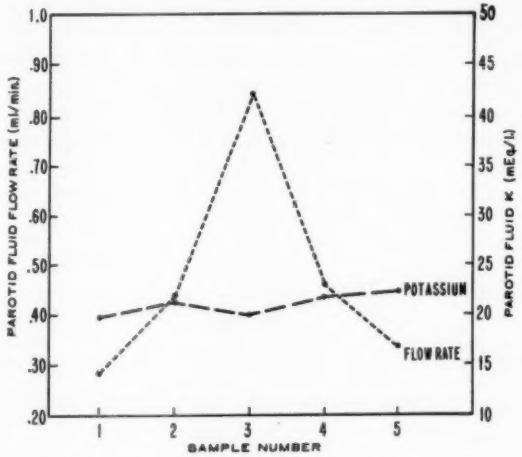

Fig. 7.—The correlation between flow rate and potassium.

means for the five test samples were 19.80 (S.D., 4.30), 21.16 (S.D., 3.07), 20.12 (S.D., 4.44), 21.79 (S.D., 3.74), and 22.39 (S.D., 2.94) mEq. per liter. "Between-subjects" potassium (Table IV) was negatively correlated with sodium ($P < .01$) and chloride ($P < .01$) but insignificantly related to flow rate and to total protein.

TABLE III. THE AVERAGE "WITHIN-SUBJECTS" CORRELATION COEFFICIENTS FOR FIVE PAROTID FLUID SAMPLES FROM EACH OF TWENTY-FIVE MALE SUBJECTS AND THE PROBABILITY OF THEIR SIGNIFICANCE

	POTASSIUM	SODIUM	TOTAL PROTEIN	CHLORIDE
Flow rate	−0.12 ($> .05$)*	0.98 ($< .01$)	0.82 ($< .01$)	0.96 ($< .01$)
Potassium		−0.16 ($> .05$)*	0.27 ($< .05$)	−0.25 ($> .05$)
Sodium			0.85 ($< .01$)	0.95 ($< .01$)
Total protein				0.83 ($< .01$)

*The correlation between these two variables differed significantly from one subject to another.

TABLE IV. THE "BETWEEN-SUBJECTS" CORRELATION COEFFICIENTS AND THE PROBABILITY OF THEIR SIGNIFICANCE

	POTASSIUM	SODIUM	TOTAL PROTEIN	CHLORIDE
Flow rate	0.09 ($> .05$)	0.36 ($> .05$)	−0.25 ($> .05$)	0.35 ($> .05$)
Potassium		−0.71 ($< .01$)	0.25 ($> .05$)	−0.60 ($< .01$)
Sodium			−0.29 ($> .05$)	0.94 ($< .01$)
Total protein				−0.22 ($> .05$)

Sodium: Sodium was found to be strongly correlated with flow rate (Fig. 8), within subjects, in a positive fashion ($P < .01$). Significant ($P < .01$) correlations were also seen for sodium with total protein and for sodium and chloride. The sodium-potassium correlation was not significant (Table III). As stimulation intensity was increased the sodium mean rose from 9.95 (S.D., 14.56) to 20.22 (S.D., 20.27) to 49.88 (S.D., 28.43) mEq. per liter, and as intensity was decreased the mean fell to 20.20 (S.D., 17.61) and to 12.88 (S.D., 14.47) mEq. per liter. "Between-subjects" sodium (Table IV) was negatively correlated with potassium ($P < .01$) and positively correlated with chloride ($P < .01$) but insignificantly related to flow rate and total protein.

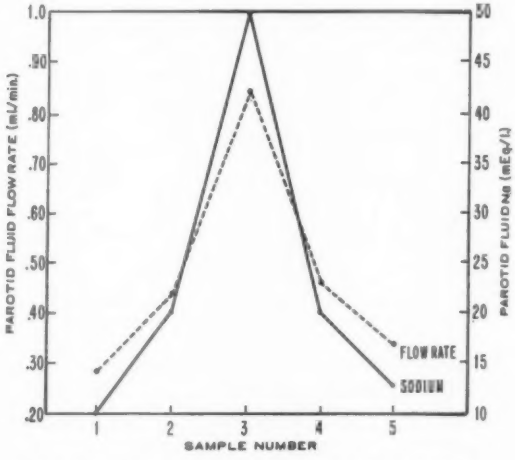

Fig. 8.—The correlation between flow rate and sodium.

Total protein: This variable was positively correlated with rate of flow (Fig. 9), sodium, and chloride ($P < .01$) within subjects. The positive correlation between total protein and potassium (Table III) was barely significant ($P < .05$). Total protein mean values for the five specimens were 132 (S.D., 56.8), 158 (S.D., 47.1), 227 (S.D., 65.2), 144 (S.D., 51.7), and 129 (S.D., 41.7) mg. per 100 ml. of parotid fluid. "Between-subjects" total protein (Table IV) was insignificantly related to flow rate, potassium, sodium, and chloride.

Chloride: Flow rate and chloride were positively correlated ($P < .01$) within subjects (Fig. 10). Chloride was also positively related to sodium

($P < .01$) and to total protein ($P < .01$). The negative correlation with potassium was not significant (Table III). As stimulus intensity was altered, the mean chloride value changed from 16.15 (S.D., 7.66) to 20.75 (S.D., 12.13), to 34.02 (S.D., 17.25), to 20.68 (S.D., 11.99), and to 17.57 (S.D., 9.01) mEq. per liter. "Between-subjects" chloride (Table IV) was negatively correlated to potassium ($P < .01$), positively correlated to sodium ($P < .01$), and insignificantly related to flow rate and to total protein.

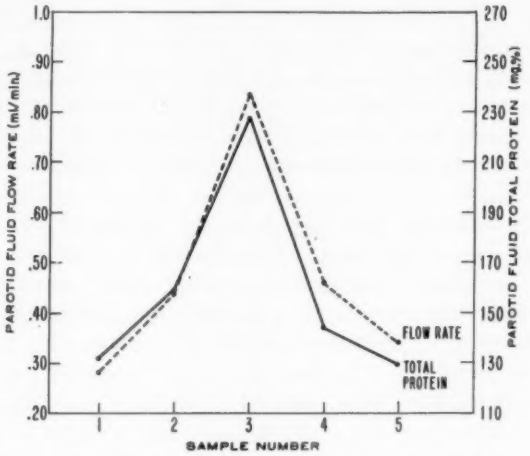

Fig. 9.—The correlation between flow rate and total protein.

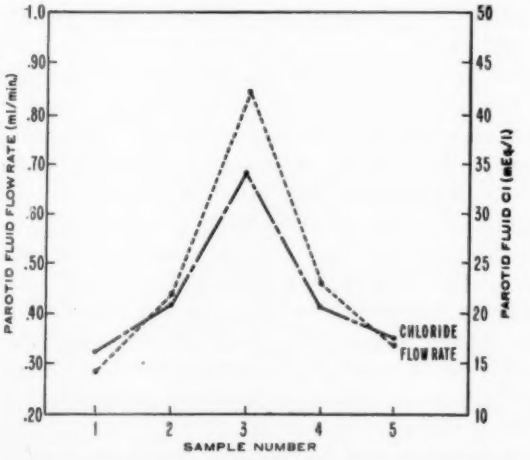

Fig. 10.—The correlation between flow rate and chloride.

The findings that significant positive correlations existed between flow rate and bicarbonate, calcium, pH, sodium, total protein, and chloride, that inorganic

phosphate was negatively correlated with rate of flow, and that potassium appeared to be independent of flow rate are in agreement with the majority of published reports.

In a study of human parotid secretion, Chauncey and associates[17] found a positive correlation between flow rate and sodium (0.50), calcium (0.38), bicarbonate (0.55), and pH (0.52). Potassium exhibited a negative correlation (–0.30). All of these coefficients were significant at less than 0.05. In this study two 12.5 ml. samples of fluid were collected from each subject on each of two successive days under identical stimulation. The mean flow rates and chemical composition are not provided, but only the first sample mean flow differed significantly from the other samples. From a statistical point of view, flow rate-concentration correlations based upon three nondiffering samples and one sample of lower flow rate from each person would be most difficult to interpret, particularly when samples from each participant were collected on different days.

Hildes and Ferguson[14] used glucose candy and dilute acetic acid as stimulants and found that human parotid fluid sodium, chloride, pH, and bicarbonate were positively correlated with flow and that potassium and phosphate concentrations were negatively correlated with flow up to a flow rate of 0.5 ml. per minute and thereafter independent of flow rate influences. Hildes,[18] in comparing the secretions of the pancreas and the parotid gland, found that sodium, bicarbonate, and chloride were positively correlated with parotid gland flow rates and that potassium and phosphate were independent of secretion rates.

Thaysen and colleagues,[19] analyzed forty-seven human parotid fluid samples collected at secretory rates of from 0.20 to 4.26 ml. per minute and found a positive flow rate effect on sodium and chloride while potassium concentration was largely independent.

The positive correlation between human parotid flow rate and chloride concentration has been confirmed by Carter and co-workers[20] and by Bramkamp,[21] while Coats and Wright[22] could find no clear relationship between parotid flow rate and chloride concentration in sheep. Ferguson and associates[23] found human parotid flow rate and potassium concentration to be negatively correlated, while in dogs Langley and associates[24] determined a negative correlation between these two variables and Burgen[25] concluded that potassium was secreted from the dog parotid gland in two distinct phases: (1) a transient high rate of potassium secretion immediately upon activation of the previously inactive gland followed by (2) the establishment of a steady state when potassium became independent of flow rate. A negative flow rate-potassium relationship was also found for sheep parotid fluid.[22] Englander and co-workers[26] found human parotid fluid pH to vary directly with flow rates, but deBeer and Wilson[27] found no flow rate-pH correlation in dog parotid fluid collected under pilocarpine stimulation. The positive correlation between parotid flow rate and sodium has been confirmed in animal studies.[22, 24] Bicarbonate has been found to be positively correlated with parotid flow in experimental animals,

while phosphate was found to decrease with increases in flow rate.[22] Bramkamp[28] found protein nitrogen in human parotid fluid to increase progressively with increased secretion, but Ferguson and colleagues[23] found that, at rates of flow exceeding 0.1 ml. per minute, the protein concentration of human parotid secretion was not related to rate of flow.

SUMMARY

Five parotid fluid accommodation samples and five test samples were collected from each of fifty-three healthy young adult male subjects in two separate experiments directed toward the determination of flow rate-concentration relationships. A reproducible ascending-decending flow rate pattern was obtained by using rubber bands, gum base, sugared gum, gum base, and rubber bands for stimulation, in that order, for the collection of the specimens. "Within-subject" and "between-subject" correlation coefficients and their probability of significance were calculated for all variables in each experiment.

Positive flow rate correlations, significant at the 1 per cent level, were found for bicarbonate, calcium, pH, sodium, total protein, and chloride. An equally significant negative correlation was found between flow rate and inorganic phosphate. Potassium and flow rate were not significantly correlated.

The authors express their appreciation to Mr. Richard McNee and Mr. Al Rahe for the statistical analysis and to medical technicians J. F. McAnear, E. D. Dinger, L. Crawford, D. Weise, and E. Espinosa for technical assistance.

REFERENCES

1. Van Slyke, D. D.: Determination of the Bicarbonate Concentration of the Blood and Plasma, J. Biol. Chem. 52: 495, 1922.
2. Shannon, I. L., and Prigmore, J. R.: The Use of the Autoanalyzer in Studies of the Physiological Chemistry of the Parotid Gland, Ann. New York Acad. Sc. 87: 745, 1960.
3. Shannon, I. L., and Prigmore, J. R.: Whole Saliva Volume, Sodium, Potassium, and Chloride—Day-to-Day Comparisons Under Identical and Diverse Forms of Stimulation, J. D. Res. 38: 843, 1959.
4. Gornall, A. G., Bardawill, C. J., and David, M. M.: Determination of Serum Proteins by Means of the Biuret Reaction, J. Biol. Chem. 177: 751, 1949.
5. Cotlove, E., Trantham, H. V., and Bowman, R. L.: An Instrument and Method for Automatic, Rapid, Accurate, and Sensitive Titration of Chloride in Biological Samples, J. Lab. & Clin. Med. 51: 461, 1958.
6. Shannon, I. L.: Salivary Sodium, Potassium, and Chloride Levels in Subjects Classified as to Dental Caries Experience, J. D. Res. 37: 401, 1958.
7. Shannon, I. L., and Prigmore, J. R.: Physiologic Chloride Levels in Human Whole Saliva, Proc. Soc. Exper. Biol. & Med. 97: 825, 1958.
8. Shannon, I. L.: Physiologic Chloride Levels in Human Whole Saliva Elicited by Rubber Band Stimulation, School of Aviation Medicine, USAF, Report No. 57-142, August, 1957.
9. Shannon, I. L.: Physiologic Chloride Levels in Human Whole Saliva Collected Without Exogenous Stimulation, School of Aviation Medicine, USAF, Report No. 58-26, January, 1958.
10. Shannon, I. L., and Prigmore, J. R.: A search for Diurnal Characteristics in Human Whole Stimulated Saliva, School of Aviation Medicine, USAF, Report No. 59-46, June, 1959.
11. Shannon, I. L.: Whole Saliva Volume, Sodium, Potassium and Chloride—Day-to-Day Comparisons Under Identical and Diverse Forms of Stimulation, School of Aviation Medicine, USAF, Report No. 58-83, April, 1958.

12. Shannon, I. L., and Prigmore, J. R.: Evaluation of the Van Slyke and Hiller Modification of the Method of Sendroy as Applied to the Chloride Analysis of Saliva, School of Aviation Medicine, USAF, Report No. 58-78, May, 1958.
13. White, A. G., Entmacher, P. S., Rubin, G., and Leither, L.: Physiological and Pharmacological Regulation of Human Salivary Electrolyte Concentrations: With a Discussion of Electrolyte Concentrations of Some Other Exocrine Secretions, J. Clin. Invest. **34**: 246, 1955.
14. Hildes, J. A., and Ferguson, M. H.: The Concentration of Electrolytes in Normal Human Saliva, Canad. J. Biochem. & Physiol. **33**: 217, 1955.
15. Van Slyke, D. D., and Hiller, A.: Application of Sendroy's Iodometric Chloride Titration of Protein-Containing Fluids, J. Biol. Chem. **167**: 107, 1947.
16. Sendroy, J., Jr.: Microdetermination of Chloride in Biological Fluids, With Solid Silver Iodate. II. Titrimetric Analysis, J. Biol. Chem. **120**: 405, 1937.
17. Chauncey, H. H., Lisanti, V. F., and Winer, R. A.: Human Parotid Gland Secretion: Flow Rate and Interrelationships of pH and Inorganic Components, Proc. Soc. Exper. Biol. & Med. **97**: 539, 1958.
18. Hildes, J. A.: Glandular Secretion of Electrolytes, Canad. J. Biochem. & Physiol. **33**: 481, 1955.
19. Thaysen, J. H., Thorn, N. A., and Schwartz, I. L.: Excretion of Sodium, Potassium, Chloride, and Carbon Dioxide in Human Parotid Saliva, Am. J. Physiol. **178**: 155, 1954.
20. Carter, W. J., Englander, H. R., and Weber, T. B.: Chloride Levels in Parotid Secretion, U. S. Navy Research Report NM 75 02 07, Dental Research Facility, Great Lakes, Illinois, April, 1958.
21. Bramkamp, R. G.: Urea and Chlorides in the Human Parotid Saliva, J. Lab. & Clin. Med. **22**: 677, 1937.
22. Coats, D. A., and Wright, R. D.: Secretion by the Parotid Gland of the Sheep: The Relationship Between Salivary Flow and Composition, J. Physiol. **135**: 611, 1957.
23. Ferguson, M. H., Krahn, H. P., and Hildes, J. A.: Parotid Secretion of Protein in Man, Canad. J. Biochem. & Physiol. **36**: 1001, 1958.
24. Langley, L. L., Gunthorpe, C. H., and Beall, W. A.: Parotid Clearance of Sodium and Potassium, Am. J. Physiol. **195**: 693, 1958.
25. Burgen, A. S. V.: The Secretion of Potassium in Saliva, J. Physiol. **132**: 20, 1956.
26. Englander, H. R., Mau, L. M., Hoerman, K. C., and Chauncey, H. H.: Dental Caries Activity and the pH, Titratable Alkalinity, and Rate of Flow of Human Parotid Saliva, J. D. Res. **37**: 906, 1958.
27. DeBeer, E. J., and Wilson, D. W.: The Inorganic Composition of the Parotid Saliva of the Dog and Its Relation to the Composition of the Serum, J. Biol. Chem. **95**: 671, 1932.
28. Bramkamp, R. G.: The Protein Content of Human Parotid Saliva, J. Biol. Chem. **114**: 369, 1956.

HISTOCHEMICAL STUDY OF CYTOCHROME OXIDASE IN NORMAL AND INFLAMED GINGIVA

M. S. Burstone, D.D.S., Bethesda, Md.

INTRODUCTION

Cytochrome oxidase is a major respiratory enzyme which has been implicated in the metabolism of mammalian tissues.[1] Most of the studies on this enzyme have employed biochemical techniques. With regard to the oral tissues, biochemical determinations of this enzyme have been made in salivary glands, dental pulp, and gingiva.[2, 3] Cytochrome oxidase activity of human gingiva has been found to be rather low as compared with that of most other tissues.[3] Of interest is the observation that there is an increased oxygen consumption of inflamed gingiva.[4, 5]

Since gingiva is a nonhomogeneous tissue containing a number of different cell types, a histochemical approach was deemed advisable in order that the exact cellular sites of activity in both health and disease could be visualized. Until recently histochemical demonstration of cytochrome oxidase has been rather unsatisfactory, since the so-called "nadi" procedure is prone to diffusion artifacts and dye deterioration. Within the last few years more definitive histochemical techniques have been developed, which give very good histologic localizations of cytochrome oxidase.[1] With these techniques, new histologic findings on the cellular sites of cytochrome oxidase in normal and inflamed gingiva were obtained.

METHODS

Gingival specimens were obtained from adult patients with chronic periodontitis. These specimens also included "normal" adjacent gingiva. In addition, interdental papillae were obtained from patients with normal gingiva at the time of extraction of teeth.

Tissues were frozen and sectioned as previously described.[1] The sections, which were 8 to 10 microns in thickness, were incubated in substrates containing p-aminodiphenylamine and one of the following couplers[1]:

A. 8-amino-1,2,3,4-tetrahydroquinoline
B. 3 amino-9-ethyl carbazole
C. p-methoxy-p-aminodiphenylamine

From the National Institute of Dental Research, National Institutes of Health, United States Public Health Service, Department of Health, Education, and Welfare.

Representative sections were incubated in substrate solutions with and without cytochrome *c*.* The incubation periods ranged from one to two hours. Following incubation, sections were treated with cobalt-formalin as previously described.[1]

OBSERVATIONS

All substrates gave essentially identical localizations. The staining reaction was found to be markedly augmented by cytochrome *c*. The attached and free normal gingiva up to the crevicular epithelium revealed little or no reaction. When staining occurred, it was primarily delineated to the basal-cell layer (Fig. 1). The underlying lamina propria exhibited activity of fibroblasts. The cells of the crevicular epithelium and epithelial attachment exhibited a distinct contrast in activity as compared with those of the adjacent free gingiva (Fig. 2).

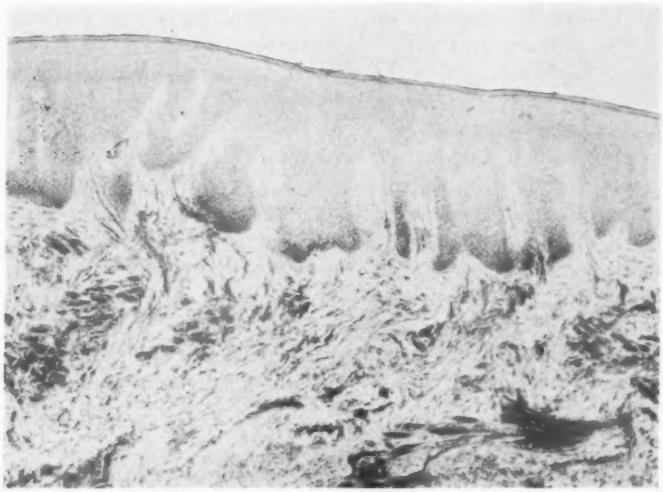

Fig. 1.—Normal human attached gingiva incubated in the presence of cytochrome *c*. Note activity of basal-cell layer and underlying connective tissue. (Magnification, ×73.)

In all cases of gingival inflammation an increased staining reaction of the crevicular epithelium and epithelial attachment was observed. In addition, distinct and characteristic activity was found in the adjacent free gingiva up to the free gingival groove (Fig. 3). The free gingival groove appeared to represent a distinct zone of demarcation of enzyme activity. Although the staining of the underlying lamina propria was variable, there was nevertheless a distinct increase in activity as compared with normal controls (Figs. 4 and 5). Lymphocytes and plasma cells were also found to be stained.

*Sigma Chemical Company, St. Louis, Missouri.

DISCUSSION

All living mammalian cells are known to contain the so-called cytochrome systems, which are involved in electron-transport and oxidative mechanisms. In this series we find cytochrome oxidase, a major respiratory enzyme. Muscle and central nervous system tissues, for example, are known to exhibit high

Fig. 2.—Normal human gingiva showing activity of crevicular epithelium and epithelial attachment. (Magnification, ×73.)

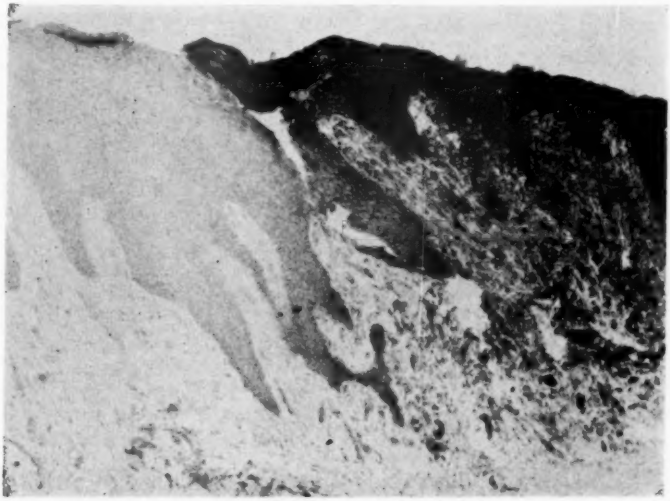

Fig. 3.—Human gingiva (gingivitis) showing marked increase in oxidase activity associated with inflammation. (Magnification, ×73.)

cytochrome oxidase activity.[1] Of interest is the observation that the respiration of human skin is only a fraction of that of brain or muscle. It has been suggested that the main steps in energy metabolism of the skin (and presumably the gingiva) proceed as in other tissues—by glycolyses of carbohydrates and finally by a transfer of electrons through the cytochrome system to molecular oxygen.[6]

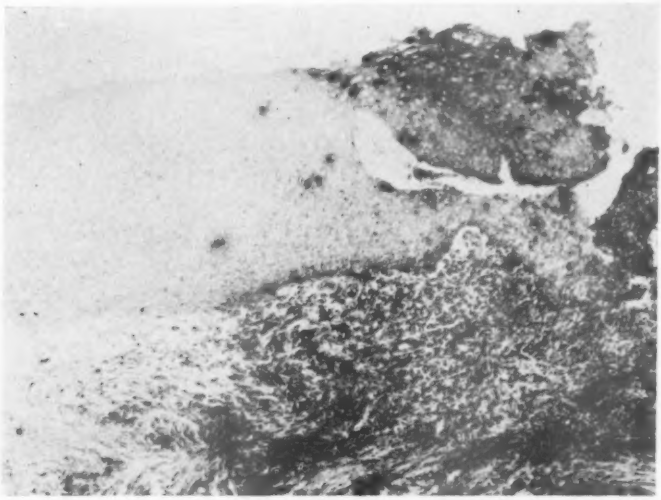

Fig. 4.—Area similar to that shown in Fig. 3. Note increased stromal reaction. (Magnification, ×73.)

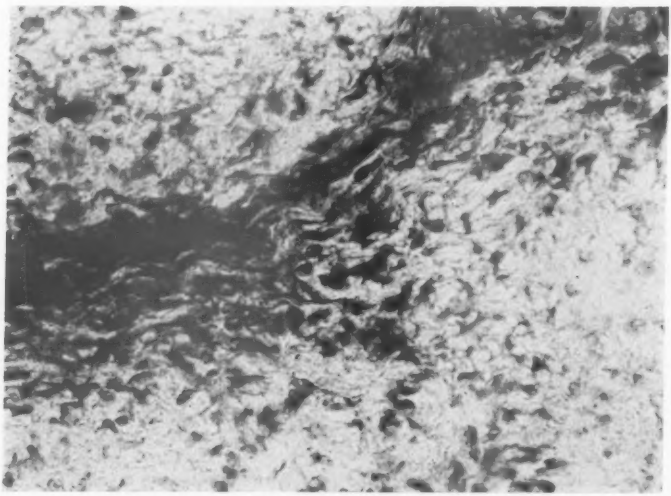

Fig. 5.—Higher magnification of area of lamina propria shown in Fig. 4, showing activity of fibroblasts. (Magnification, ×285.)

In 1926 Warburg[7] found that there was an increased oxygen consumption associated with tissue damage. Glickman and his colleagues,[4] in their significant study, were the first to demonstrate that an increased oxygen consumption was associated with gingivitis. They assumed that the increased oxygen consumption in gingivitis was related to the inflammatory infiltrate and connective tissue proliferation. These findings were further confirmed and elaborated upon by Schrader and Schrader.[5] Quantitative determination of cytochrome oxidase in normal rabbit gingiva has been reported by Eichel and Swanson.[3] However, there do not appear to be any quantitative or histochemical studies of changes in cytochrome oxidase in gingivitis.

In quantitative determinations the question of the exact sites of oxidase activity at the histologic and cytologic levels remains unanswered. These sites, of course, were definitively elucidated by the present study. Of particular interest is the new finding of increased cytochrome oxidase of the epithelial cells per se. Because of the significance of cytochrome oxidase in tissue metabolism, histochemical study of its distribution warrants study as a possible index of the effectiveness of therapeutic procedures in the treatment of periodontal disease.

CONCLUSIONS

Definitive histochemical localizations of cytochrome oxidase in normal and chronically inflamed gingiva were obtained by new histochemical techniques.

The cytochrome oxidase activity of normal gingiva is low and is observed in the crevicular epithelium, the epithelial attachment, and the basal-cell layer of the free and attached gingiva.

In chronic gingivitis, a striking increase in activity is manifested in the epithelium from the free gingival groove through the epithelial attachment. The underlying connective tissue stroma exhibits a variable increase in activity.

REFERENCES

1. Burstone, M. S.: Histochemical Demonstration of Cytochrome Oxidase With New Amine Reagents, J. Histochem. & Cytochem. **8**: 63, 1960.
2. Person, P.: Dynamic Equilibria of Oral Tissues. II. Cytochrome Oxidase and Succinoxidase Activity of Oral Tissues, J. Periodont. **26**: 99, 1955.
3. Eichel, B., and Swanson, A. A.: Oxidative Enzymes. 1. Succinic Dehydrogenase, DPN Cytochrome *c* Reductase, Cytochrome Oxidase, and Catalase in Oral, Liver, and Brain Cortex Tissues, J. D. Res. **36**: 581, 1957.
4. Glickman, I., Turesky, S., and Hill, R.: Determination of Oxygen Consumption in Normal and Inflamed Human Gingiva Using the Warburg Manometric Technic, J. D. Res. **28**: 83, 1949.
5. Schrader, H. K., and Schrader, R.: Oxygen Uptake by Normal and Inflamed Gingiva and Saliva, Helvet. odont. acta **1**: 13, 1959.
6. Rothman, S.: Physiology and Biochemistry of the Skin, Chicago, 1954, University of Chicago Press.
7. Warburg, O.: Der Staffwechsel der Tumoren, Berlin, 1926, Julius Springer.

ANNOUNCEMENTS

Armed Forces Institute of Pathology

The Institute will present its annual course, "Pathology of the Oral Regions," April 10 to 14, 1961, in Washington, D. C.

The five-day course will emphasize pathology of the oral mucosa and jaws as well as oral manifestations of systemic disease. There are no fees for the program.

Quotas have been established for both civilian and military personnel. Members of the Federal Services should apply through their respective services. Civilians interested in enrolling in the course may apply to the Director, Armed Forces Institute of Pathology, Washington 25, D. C.

American Academy of Dental Medicine Announces 1961 Cruise Convention

The Academy will conduct its 1961 meeting in the form of a cruise to Bermuda and Nassau aboard the luxury liner S. S. Ocean Monarch from Friday, May 26, to Saturday, June 3. The ship is completely air-conditioned, and 157 outside rooms provide private facilities. Rates range from $200.00 per person, all-inclusive.

The scientific discussion will feature the topic "Recent Trends in Dental Medicine." A joint meeting with the Bermuda Dental Society at that island community will feature the program with Dr. Hermann Becks, Academy president, as a speaker.

Members are urged to make reservations early by sending a deposit of $100.00 to Travel Guide Agency, attention of Mr. Eugene Fisher, 416 North Charles St., Baltimore, Maryland.

Registration fee for the scientific session is $10.00. Checks should be made payable to Dr. Rudy Palermo, 106 Central Ave., Brooklyn, New York.

Illinois Section of the American Dental Society of Anesthesiology

The Society is offering a one-day program entitled "Recent Advances in Dental Anesthesia" on Feb. 5, 1961. This will be held at the Conrad Hilton Hotel immediately preceding the Mid-Winter Meeting of the Chicago Dental Society. Participating in the program will be Drs. Morgan Allison, Professor of Oral Surgery and Anesthesiology, Ohio State University; Edward Dobbs, Professor of Pharmacology, University of Maryland; Leonard Monheim, Professor of Anesthesiology, University of Pittsburgh; and Stanley Spiro, Clinical Assistant Professor of Anesthesiology, Columbia University.

Further information may be secured from Dr. E. Stuebner, 808 South Wood St., Chicago 12, Illinois.

Bicentennial of Pierre Fauchard's Death

Next year will mark the 200th anniversary of the death of Pierre Fauchard, known as the "Creator of Modern Dentistry." French dentists have decided to observe the event

with éclat and cordially invite their confreres of all countries to join them. There will be a meeting in the amphitheater of the Sorbonne in the presence of members of the French Government. Fauchard's *Le Chirurgien Dentiste*, an immortal publication, will be re-edited, and a postage stamp bearing Fauchard's portrait probably will be issued. The meeting will be held in conjunction with the Paris congress of the American Dental Society of Europe (June 24 to 30, 1961) and the annual reunion of the Fédération Dentaire Internationale in Helsinki, Finland (July 9 to 16, 1961).

Inquiries should be addressed to M. Max Filderman, Secrétaire-Général du Comite National du Bicentenaire de Fauchard, 67, rue de Tocqueville, Paris XVIIe, France.

Internships and Residency at Grand Central Hospital

Two rotating internships will be available in July, 1961, at Grand Central Hospital in New York City. Internship includes training in prosthetics, root canal, orthodontics, crown and bridge, maxillofacial prosthetics, general anesthesia, operative dentistry under general anesthesia, exodontia, and oral surgery with rotation to Morrisania City Hospital. The department is certified by the American Dental Association.

An anesthesia residency for a dentist will be available at Grand Central Hospital in July, 1961. A background of internship is required.

Application should be made to Dr. Bernard M. Cohen, Director, Department of Dentistry, Grand Central Hospital, 321 East 42nd St., New York, New York.

University of Pennsylvania

The School of Dentistry announces the following postgraduate courses:

Dentistry for Handicapped Children. Manuel M. Album and associates. Dec. 12 to 14, 1960.

Seminar on Handicapped Problems. Manuel M. Album and associates. Dec. 15, 1960. (For persons who have attended a postgraduate course on dentistry for handicapped children.)

Hypnosis Applied to Dentistry. William T. Heron. Jan. 16 to 18, 1961.

Prosthetic Relations of the Temporomandibular Joint. Ernest R. Granger and associates. Jan. 16 to 20, 1961.

Impacted Teeth. Victor H. Frank and associates. Jan. 25 to 27, 1961.

Cephalometry. Viken Sassouni. Jan. 30 to Feb. 1, 1961.

Applied Anatomy. Harry Sicher. Feb. 22 to 24, 1961.

Partial Dentures. M. M. DeVan and associates. Feb. 27 to March 3, 1961.

For further information, please write to Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

Research and Teaching Traineeships at University of Pennsylvania

Research and teaching traineeships in the basic sciences, in periodontology, and in the comprehensive treatment and rehabilitation of the cleft palate patient are available at the School of Dentistry, University of Pennsylvania.

Candidates for the traineeships in the basic sciences must qualify for admission to the Graduate School of Arts and Sciences. These traineeships may lead to M.S. or PH.D. degrees.

All traineeships are supported by the United States Public Health Service. Annual stipends are $4,500.00 and up, depending on the qualifications and needs of the successful candidates. Inquiries regarding the basic science and cleft palate traineeships should be

sent to the Chairman, Committee on Traineeships and Fellowships, University of Pennsylvania, School of Dentistry, 4001 Spruce St., Philadelphia 4, Pennsylvania. Inquiries regarding the traineeships in periodontology should be sent to the Dean, Graduate School of Medicine, 237 Medical Laboratories, University of Pennsylvania, Philadelphia 4, Pennsylvania.

Temple University

The School of Dentistry announces the following graduate and postgraduate courses for 1960-61:

High Speed in Operative Dentistry. Carlos Weil and Charles A. Nagle, Jr. Dec. 7, 1960, and March 1, 1961. Tuition, $35.00. Enrollment limited to ten.

Implant Dentures. Aaron Gershkoff and Norman I. Goldberg. Jan. 9 to 13, 1961. Tuition, $200.00.

Anatomy—Head and Neck Dissection. Richard M. Snodgrasse. Feb. 1 to May 17, 1961 (fifteen Wednesdays, from 1 to 5 P.M.). Tuition, $300.00. Enrollment limited to six.

Advanced Fixed Prosthesis. George Straussberg. Feb. 6 to 11, 1961. Tuition, $200.00. Enrollment limited.

Present-Day Periodontal Practice. Jacoby T. Rothner, Irving Glickman, Bernard Saturen, and Neal W. Chilton. Feb. 14 to April 18, 1961 (each Tuesday for ten weeks). Tuition, $250.00. Enrollment limited to ten.

Practice Administration. Jay H. Eshleman. Feb. 18, 1961. Tuition, $25.00. Enrollment limited to twenty.

Periodontal Prosthesis. Morton Amsterdam, D. Walter Cohen, and associates. Feb. 20 to 24, 1961. Tuition, $275.00. Enrollment limited.

Electronic Electrosurgery. Maurice J. Oringer. March 3 and 4, 1961. Tuition, $60.00. Enrollment limited.

Immediate Surgical Endodontics. Leonard Parris, Harold Rappaport, and Robert Uchin. March 6 to 11, 1961. Tuition, $175.00. Enrollment limited to eight.

Advanced Surgical Techniques in Periodontal Therapy. Saul Schluger and Jack Alloy. March 25 to 29, 1961. Tuition, $300.00. Enrollment limited to ten.

Full Denture Prosthesis. Bernard Jankelson and associates. May 8 to 12, 1961. Tuition, $200.00. Enrollment limited.

Graduate Orthodontics. Robert B. Hedges and associates. Sept. 7, 1961 (a two-year course). Tuition $450.00 per semester.

For application and additional information, write to Dr. Louis Herman, Director of Postgraduate Studies, Temple University School of Dentistry, 3223 North Broad St., Philadelphia 40, Pennsylvania.

Tufts University

The School of Dental Medicine announces that two fellowships in periodontology intended for graduate dentists interested in research and teaching careers in periodontology are now available. In addition to participation in an extensive research program, the fellowships offer training in periodontal pathology and an opportunity to prepare for advanced degrees. There is an annual stipend of $5,000.00, and the fellowships are renewable.

Additional information and application may be obtained from Dr. Irving Glickman, Periodontology, 136 Harrison Ave., Boston 11, Massachusetts.

Medical College of Virginia

Fellowships will be available to the 1960-1961 session, leading to M.S. or Ph.D. degrees in basic sciences. These fellowships are supported by funds from the National Institute of Dental Research, the United States Public Health Service, and will allow a stipend of $5,500.00 for dental graduates. Qualified applicants will be considered for degrees in anatomy, chemistry, microbiology, pathology, and pharmacology. According to the wishes of the trainees, a reasonable amount of clinical training may be included.

Further information can be obtained from the Director of Graduate and Postgraduate Studies, School of Dentistry, Medical College of Virginia, Richmond, Virginia.

University of Washington School of Dentistry

The Graduate School of the University of Washington, in conjunction with the facilities of King County Hospital and University of Washington Hospital, is presently offering three years of graduate educatica leading to a master's degree in oral surgery. The nature and scope of this program qualifies the trainee for certification by the American Board of Oral Surgery.

One student is selected annually to enter the academic year of basic sciences and research given at the University. The second and third years are spent in the King County Hospital residency. This training includes, besides oral surgery, general anesthesia, pathology, and radiology.

There is no University of Washington stipend for the academic year, but the Oral Surgery Department will assist wherever possible in obtaining a United States Public Health Service grant to carry out the graduate student research. The junior resident's salary is $150.00 a month plus board, room, and laundry, while the senior resident receives the same benefits with $200.00 a month.

For further information, please write Dr. John D. Gehrig, Associate Professor, Executive Officer, School of Dentistry, University of Washington, Seattle 5, Washington.

American Association of Endodontists

The Association is sponsoring a symposium entitled "Biology of the Dental Pulp," which will be presented on Thursday, Feb. 2, 1961, at the Conrad Hilton Hotel in Chicago, Illinois.

The fee for the entire day, including lunch with the clinicians, is $25.00. Checks, made payable to the Association, should be forwarded to Dr. L. Parris, 1930 Chestnut St., Philadelphia, Pennsylvania.

REVIEWS OF THE LITERATURE

BOOK REVIEWS

Nitrous Oxide in Dentistry—Its Dangers and Alternatives. J. G. Bourne. Chicago, 1960, The Year Book Publishers, Inc. Price, $6.00.

As the title indicates, this book is concerned with the shortcomings of nitrous oxide anesthesia in dentistry and the possibility of an alternative inhalation anesthetic agent. The difficulty in administering nitrous oxide and the possible dangers of oxygen restriction motivated the author's research in this area. Inquiry revealed that damage to cerebral function had been reported following nitrous oxide, and investigation of cerebral circulation during nitrous oxide anesthesia uncovered evidence of the occurrence of fainting spells during the induction phases. The author limited his research primarily to ambulatory patients in dental surgery, feeling that the greatest dangers of nitrous oxide anesthesia were present in its use with this type of patient because most patients are anesthetized in a sitting-up position in which fainting would lead to cerebral stagnation and hence to postoperative impairment of cerebral function. The author's investigations were also largely limited to cases in which nitrous oxide was used as the induction agent. Dr. Bourne's studies led him to conclude that nitrous oxide had numerous shortcomings, and he proposes cyclopropane as an alternative anesthetic agent for use in dentistry. He suggests methods for eliminating or reducing the hazards in the use of cyclopropane in dentistry, such as the use of an unignitable mixture of 50 per cent cyclopropane, 25 per cent nitrous oxide, and 25 per cent oxygen. His method of a preset mixture in a closed breathing system seems to meet the basic requirements of simplicity, effectiveness, and safety and may be well worth further investigation. In addition to an investigation of nitrous oxide anesthesia and a new approach to dental anesthesia, the book also includes discussions of nitrous oxide potency and of cyclopropane and its clinical trials; a note on fainting under nitrous oxide anesthesia; and several case reports of the after effects of nitrous oxide anesthesia.

Vincent D. Palumbo.

Atlas of Clinical Pathology of the Oral Mucous Membrane. Baliant J. Orban, Professor of Periodontics, Loyola University School of Dentistry, Chicago, Illinois, and Frank M. Wentz, Chairman, Department of Periodontics, Loyola University School of Dentistry, Chicago, Illinois. Second edition. St. Louis, 1960, The C. V. Mosby Company. Price, $14.50.

This book by Orban and Wentz, with forty-five contributors, was well received in its first appearance. It contains a thorough and complete outline of diseases of the oral cavity. Each clinical photograph, in color, is accompanied by an explanation of the pathologic process, which should be of great value to the student. The book is recommended to the dentist and the physician.

Thomas J. Cook.

Noyes' Oral Histology and Embryology—With Laboratory Directions. Isaac Schour, B.S., D.D.S., M.S., Ph.D., Sc.D., Professor of Histology and Head of Department and Dean, University of Illinois, College of Dentistry, Chicago, Illinois. Eighth edition. Philadelphia, 1960, Lea & Febiger. Price, $11.50.

Changes in and additions to the text matter have resulted in a complete resetting of the book for the eighth edition. A chapter on the temporomandibular joints has been added, and comparative and experimental dental histology are discussed more fully, each in a separate chapter. Findings made possible by the improved use of electron microscopy, histochemistry, and other recent developments in biologic and physical methods are included, as are the results of recent research in experimental tooth development. A well-considered chapter rearrangement increases the already effective presentation of the subject. Similarly, the chapter on laboratory exercises has been improved by a reorganization and shortening of the material that it contains. Three new color plates and forty black and white illustrations have been added.

Edmund I. Parnes.

Minor Tooth Movement in General Practice. Leonard Hirschfeld, B.A., D.D.S., Associate Professor of Dentistry, Section of Periodontology, School of Dental and Oral Surgery, Columbia University, New York, New York. St. Louis, 1960, The C. V. Mosby Company. Price, $12.00.

This is a textbook for all concerned with generalized malocclusions and particularly for the periodontist and orthodontist. However, it will find wide use among dentists in general practice. The general purpose of this book is to provide information which can help furnish answers to the following basic questions about a given case: (1) Is there a malposition of one or several teeth in this case? (2) What has caused the malposition? (3) Can I, as a general practitioner, correct it? (4) Should I attempt to correct it? (5) If so, what method should I use? (6) How shall I proceed? (7) What must I do to make the tooth stay in its correct position after it has been moved?

There are 332 pages with 293 illustrations. The line drawings, reproductions of x-rays, and photographs are excellently prepared.

Edmund I. Parnes.

Dental Practice Administration. Robert K. Stinoff, D.D.S., Akron, Ohio. St. Louis, 1960, The C. V. Mosby Company. Price, $7.50.

I do not know the author of this book or anything about him. After reading the text, however, it is my privilege to assume that Dr. Stinoff made a success of his dental practice.

Many times I have heard a young graduate say: "We had nothing whatsoever about the business side of practice in our dental school." The young dentists learn of locations, what to purchase, how to pay for it, and the running of the office from the detail man.

This book will answer many of the questions that the new graduate asks and prove to others that they could make better use of their time.

It is recommended to the senior student, to the new graduate, and to those who would like some new ideas.

Thomas J. Cook.

Bibliography on Saliva. Compiled by Bibliography Section, Science and Technology Division, Library of Congress. Price, $3.00.

This bibliography on saliva, sponsored by The Office of Naval Research, is on sale by the United States Government Printing Office. It is a guide to the existing literature with complete coverage through 1957, with additional coverage of specific journals through

1958. This 447 page text is recommended to the dental profession and may be obtained from the Superintendent of Documents, United States Government Printing Office, Washington 25, D. C.

Thomas J. Cook.

Allergie und Zahnmedizin (Allergy and Dental Medicine). Prof. Dr. med. Max Spreng, University of Basel. Leipzig, 1959, Johann Ambrosius Barth. 137 pages, 84 illustrations.

This monograph deals with fundamental knowledge of allergy as well as the causes of allergic reactions in the mouth, whether they are of a general nature or due to foreign material in the mouth, such as metal combinations and synthetic materials. Tests are described, and treatment is indicated.

Kurt H. Thoma.

ABSTRACTS OF CURRENT LITERATURE

Exfoliative Cytology in Radiotherapy of Oral Cancer. W. Umiker, R. Rapp, I. Lampe, and H. B. Latourette. Radiology 75: 107, July, 1960.

The exact biologic status of an oral carcinoma is often difficult to assess on the completion of irradiation. As immediate biopsy is not advisable, oral smears may be of value in the diagnosis of residual neoplasm.

A study was made of twenty-five cases in which direct smears were taken from the sites of irradiated oral carcinomas of the floor of the mouth, palate, gingiva, tongue, tonsil, and glossopalatine fold.

Smears taken at the completion of radiation therapy were of little or no practical value in determining the presence of residual neoplasm. Smears taken during the first few weeks after treatment correlated well with the clinical course and biopsy findings.

Oral cytology provides a simple, painless, rapid, and reliable tool in the follow-up study of irradiated oral carcinomas.

Edmund I. Parnes.

Facial Pain. A. P. Friedman, C. A. Carton, and A. Hirano: Postgrad. Med. 27: 756, June, 1960.

The underlying disorder in facial pain may be anatomic, physiologic, or psychologic. Precise diagnosis and treatment of facial pain usually are quite difficult because of the complex cranial and cervical innervation, the intricate vascular supply, and the possible emotional modulation of response to a painful stimulus.

The authors present an excellently illustrated, concise, rational outline of the various categories of facial pain.

Facial pain is grouped into three main categories: (1) typical neuralgias, primarily neurogenic; (2) atypical neuralgias, primarily vascular and psychogenic; and (3) facial pain secondary to other extracranial and intracranial causes.

In discussing psychogenic pain, the authors state that it "cannot be distinguished from pain of physical origin; both are 'real.' Most pain has a physiologic basis, but neurotic mechanisms exaggerate the suffering."

Edmund I. Parnes.

Induction of Anesthesia in Young Children. R. J. Hamer Hodges. Lancet 1: 82, 1960.

A procedure directed toward lessening the general psychic trauma of surgery and anesthesia in young children is presented.

A group of 142 patients, aged 3.5 to 9.5 years, who underwent tonsillectomy and adenoidectomy, is reported. Twenty-four hours before operation the patients were admitted to a special ward and treated as a group. The preoperative ritual was explained and demonstrated. Atropine alone was used as premedication. Thiopentone was given intravenously to induce anesthesia, and suxamethonium chloride was given to facilitate nasotracheal intubation. Anesthesia was maintained with thiopentone intravenously followed by 60 to 70 per cent nitrous oxide in oxygen through an endotracheal tube. As soon as possible after consciousness was regained and at least once before discharge from the hospital, patients were interviewed for reassurance and discussion. The children were seen as outpatients one month after operation.

The results demonstrate that psychic trauma is not an inherent part of anesthesia and surgery and that good results can be achieved without any premedication. This series may be useful as a base line for assessing premedicants given to children.

T. J. C.

Hypnosis as an Adjunct in Anesthesiology. A. M. Betcher. New York J. Med. 60: 816, 1960.

Children are very easy to hypnotize during the induction of anesthesia. They make good subjects because they are imaginative. The posthypnotic suggestion that they will have no pain on awakening is helpful. The disadvantage is that hypnosis is time-consuming and special training is required to combine hypnosis with chemoanesthesia.

T. J. C.

Experimental Production of Cancer With Tobacco Tar and Heat. D. G. Reddy, D. B. Reddy, and R. Ramochandra. Rao. Cancer, May, 1960.

Tar obtained from cigar smoke was used for painting the skin of laboratory mice, which were then exposed to heat. It is felt that the heat functioned as a cocarcinogen and accelerated neoplastic changes in the skin.

The study was stimulated by the fact that women in India who smoke cigars with the burning end in the mouth have a higher frequency of palatal cancer than men who smoke in the normal way.

T. J. C.

The Treatment of Leukoplakia Buccalis and Related Lesions With Estrogenic Hormone. I. T. Nathanson and D. B. Weisberger. New England J. Med. 221: 556, October, 1959.

Evidence is presented which suggests that leukoplakia buccalis and similar lesions are associated with alterations in the menstrual cycle in women and with a deficiency or disturbed metabolism of the sex hormones in both sexes. The authors report an improvement in the majority of cases which were treated with estrogen and add: "It is suggested that this type of therapy in combination with other well-recognized procedures may prove of value in the treatment of leukoplakia buccalis."

T. J. C.

Spontaneous Regeneration of the Seventh Nerve. A. S. James, M. Karlon, D. L. Kinsey, and J. M. Meagher. Arch. Surg. 81: 223, August, 1960.

This interesting experimental study was done on dogs. The right facial nerve was exposed at its point of emergence from the stylomastoid foramen to its major peripheral divisions. A 3 cm. arc of the main trunk and peripheral division was excised. The first noticeable defect was the inability to blink on the side operated upon.

Return of facial function can occur after wide excision of the seventh nerve in dogs. The nerve has tremendous regenerative potential.

This article is recommended reading for the surgeon who handles facial bone trauma.

T. J. C.

Radical Neck Dissection for Cervical Lymph Node Metastasis of Intraoral Carcinoma. J. D. Palmer and J. Martin. Arch. Surg. **81:** 233, August, 1960.

This is a very frank discussion of prophylactic neck dissection, that is, the dissection carried out in the absence of clinically involved cervical nodes. The authors' conclusions are as follows: (1) A more vigorous attempt should be made to eradicate the primary intraoral lesion; surgical excision should be carried out whenever the functional end result is compatible with the patient's health and happiness. (2) A complete radical neck dissection should be done only when neck nodes are or become palpable. (3) A careful biopsy of the primary intraoral tumor site should be performed if and when new neck nodes appear. (4) Vigorous retreatment of the primary lesion site should be carried out when the biopsy is positive.

T. J. C.

Vaccinotherapy of Chronic Herpes Simplex. S. Leonard Rosenthal. J. D. Med. **15:** 96, April, 1960.

There has been much skepticism concerning the possibility of immunizing against herpes simplex (or the unknown virus of the aphthous theory) with smallpox vaccine.

Variation of the clinical course and consequent treatment make it convenient to divide herpes simplex into primary, recurrent, and chronic types.

In order to observe the efficacy of serial vaccination, 149 persons with chronic herpes were treated with smallpox vaccine. It was possible to observe 100 for a period of two years or more. Each patient was vaccinated by the usual technique with commercial smallpox (cowpox) vaccine once a week on the right and left arms alternately. Previous trials indicated that ten vaccinations gave the surest results. Therefore, this was the number planned.

Although the duration of chronic herpes in this study had ranged from six months to forty years, serial vaccination with smallpox vaccine gave relief for two to twelve years in 88 per cent of 100 cases.

Edmund I. Parnes.

The Role of Trauma in the Pathogenesis of Temporomandibular Joint Arthrosis. Ekrem Gozum. Ann. Otol., Rhin. & Laryng. **69:** 348, June, 1960.

This paper evaluates the relation of trauma to the mandible and its eventual effect on the temporomandibular joint.

Fifty-eight cases of fracture of the mandible and three cases of acute trauma without fracture were studied, and the patients were recalled. It was found that temporomandibular joint arthrosis was seen more often in cases of fracture of the ascending ramus and the condyle.

Trauma of different intensity produces variable changes in the joint and articular disc. Damage to the fibers of the articulating surfaces and disc, fractures of the disc or subcondyle bony surfaces, or damage to the capillaries and hemarthrosis of the joint may occur as a result of acute trauma. Destruction of the articular lining surface and exudation of the fibrin into the articular cavity diminish the elasticity of the joint surfaces and restrict the gliding motion of the joint; later arthrosis may develop.

The number of patients developing symptoms of temporomandibular arthrosis shows the importance of acute trauma and the effect that continuous trauma and, in some cases, malocclusion have on the joint, causing the disease. Considering this, the importance of open reduction in cases of comminuted fractures of the mandible with displacement is seen.

Edmund I. Parnes.

AUTHOR INDEX*

A

ACKERMAN, ALFRED A. (*See* FIELD AND ACKERMAN), 668

ADAMS, ROBERT J., AND STANMEYER, WILLIAM R. Effects of prolonged Antarctic isolation on oral and intestinal bacteria, 117

ADILMAN, HOWARD B., AND ROBINSON, ERIC R. The delayed treatment of unerupted teeth in the edentulous mouth, 918

AISENBERG, MYRON S., GARDNER, ALVIN F., AND INMAN, C. L., SR. Osteoma of the mandible, 1356

— AND INMAN, B. WALLACE. Ameloblastoma arising within a globulomaxillary cyst, 1352

ALBRIGHT, CECIL R. (*See* MCKELVEY, ALBRIGHT, AND PRAZAK), 111

ALBUM, MANUEL M. Dental use of meprobamate and promazine hydrochloride for mentally retarded and emotionally disturbed patients, 423

ALLEN, NEWTON E. Pregnancy tumor superimposed upon a nevus, 657

ANDERSON, A. GORDON (*See* ATTERBURY AND ANDERSON), 913

ARCHER, W. HARRY. Myxoma of left maxilla, 139

ARMAO, THOMAS A. Diagnosis and treatment of a styloid fracture, 1423

ARWILL, TORE. The significance of the innervation of the predentine, 743

ATTERBURY, ROBERT A. Compound comminuted fracture of the mandible, 1469

— AND ANDERSON, A. GORDON. Patient's reaction to chairside oral surgical teaching, 913

—. (*See* SCHULTZ, ATTERBURY, AND VAZIRANI), 523

ATTIE, JOSEPH N., AND BLUM, BERTRAM. Hyperparathyroidism first suspected through a giant-cell epulis, 482

B

BAER, PAUL N., AND NEWTON, WALTER L. Studies on periodontal disease in the mouse. III. The germ-free mouse and its conventional control, 1134

—, SUMNER, CHARLES F., III, AND SCIGLIANO, JOHN. Studies on a hydrogenated fat–zinc bacitracin periodontal dressing, 494

BALDINGER, S. ROBERT. (*See* ROSSMAN, KAPLOWITZ, AND BALDINGER), 361

BARILE, MICHAEL F., AND SHEINGORN, ABE. The isolation and cultivation of a filterable form resembling pleuropneumonia-like organisms from the human dental pulp, 756

BARR, JOHN H., AND BROCKMAN, MELVIN K. Radiation dosage in dental offices, 696

BENDER, I. B., SELTZER, SAMUEL, AND YERMISH, MORRIS. The incidence of bacteremia in endodontic manipulation, 353

BERGENHOLTZ, A., AND THILANDER, H. Lipoma of the oral cavity, 767

BERGMAN, GUNNAR. Studies on mineralized dental tissues. XIV. The incremental pattern of the dentine in a case of osteogenesis imperfecta, 70

BERMAN, CHARLES L. (*See* ZEGARELLI, KUTSCHER, SILVERS, BEUBE, STERN, BERMAN, AND HERLANDS), 170

BERNIER, JOSEPH L. (*See* BHASKAR AND BERNIER), 1387

—. (*See* RICKLES AND BERNIER), 282

—. (*See* STANLEY, ZANDER, ROBINSON, AND BERNIER), 329

BEUBE, FRANK E. (*See* ZEGARELLI, KUTSCHER, SILVERS, BEUBE, STERN, BERMAN, AND HERLANDS), 170

BHASKAR, SURINDAR N., AND BERNIER, JOSEPH L. Mikulicz's disease, 1387

BIDDIX, JOSEPH C. (*See* PROVENZA, BIDDIX, AND CHENG), 157

BLAKE, FRED S. (*See* BLAKE AND BLAKE), 1099

BLAKE, HARRIS, AND BLAKE, FRED S. Breast carcinoma metastatic to maxilla, 1099

BLECHMAN, HARRY. (*See* GUPTA, BLECHMAN, AND STAHL), 470

BLUM, BERTRAM. (*See* ATTIE AND BLUM), 482

BOLDEN, THEODORE E. (*See* SCHULTZ, VAZIRANI, AND BOLDEN), 387

*January, pp. 1-128; February, pp. 129-254; March, pp. 255-384; April, pp. 385-512; May, pp. 513-640; June, pp. 641-766; July, pp. 767-896; August, pp. 897-1022; September, pp. 1023-1150; October, pp. 1151-1280; November, pp. 1281-1408; December, pp. 1409-1538.

BOSCO, HAROLD F. Reconstruction of mandible following bone loss due to osteomyelitis in the line of fracture, 663

BROCKMAN, MELVIN K. (*See* BARR AND BROCKMAN), 696

BROOKS, ROBERT A. (*See* SHANNON AND BROOKS), 240

—. (*See* SHANNON, PRIGMORE, AND BROOKS), 1010

BUCHANAN, G. (*See* HELSHAM AND BUCHANAN), 844

BUNAG, CELSO A. (*See* VILLA AND BUNAG), 203

BUNYAN, JOHN. The use of hypochlorite for the control of bleeding, 1026

BURKE, ROGER J. (*See* CHAUDHRY, BURKE, AND GORLIN), 584

—. (*See* CHAUDHRY, BURKE, STOESZ, AND GORLIN), 716

BURKET, LESTER W. (*See* SHIP, MORRIS, DUROCHER, AND BURKET), 1191, 1317, 1438

BURKMAN, N. WEIR. (*See* SCHMIDT, CROWLEY, HARNER, AND BURKMAN), 80

BURNETT, GEORGE W. (*See* KOZAM AND BURNETT), 229

BURSTONE, M. S. Histochemical study of cytochrome oxidase in normal and inflamed gingiva, 1501

C

CABRINI, R. L., MAISTO, O. A., AND MANFREDI, E. E. Histochemical study of pulp healing, 868

CAHN, LESTER R. The oral pathologist looks at the practice of dentistry, 331

—. (*See* SANDLER, STAHL, CAHN, AND FREUND), 994

CALISTI, L. P. (*See* COHEN, JORESS, AND CALISTI), 1382

CAMERON, JAMES R. Oral Surgery at the crossroads—which direction? 129

CAMPBELL, J. B., SEGUIN, LUCILE, KUTSCHER, AUSTIN H., AND ZEGARELLI, EDWARD V. Activity of amphotericin B against *Candida albicans*, 1273

CARNES, JOHN C. Headache of dental origin, 1314

CHACONAS, CONSTANTINE P. Herpes zoster—a primary manifestation of chronic lymphatic leukemia, 1429

CHAUDHRY, A. P., BURKE, ROGER J., AND GORLIN, R. J. Malignant melanoma of the oral cavity, 584

—, —, STOESZ, A. B., AND GORLIN, R. J. Benign neoplastic fibro-osseous lesion of the mandible, 716

— AND GORLIN, R. J. A solitary xanthoma of the attached gingiva, 449

—, —, AND MITCHELL, DAVID F. Papillary cystadenoma of minor salivary gland origin, 452

—, —, AND MOSSER, DONN G. Carcinoma of the antrum, 269

—, —, AND REYNOLDS, D. H. Sialolithiasis of a minor salivary gland, 578

—. (*See* GORLIN AND CHAUDHRY), 257

—. (*See* GORLIN, CHAUDHRY, AND KELLN), 1233

—. (*See* KELLN, CHAUDHRY, AND GORLIN), 1245

CHAUNCEY, HOWARD H. (*See* QUINTARELLI, GORDON, AND CHAUNCEY), 875

CHENG, THOMAS C. (*See* PROVENZA, BIDDIX, AND CHENG), 157

CHERASKIN, E., AND MANSON-HING, L. The normal glucose-tolerance pattern, 819

—. (*See* MANSON-HING AND CHERASKIN), 1447

CHOUKAS, NICHOLAS C., AND SICHER, HARRY. The structure of the temporomandibular joint, 1203

CHRISTENSEN, ROBERT W. Anterior open-bite or retrognathia corrected by a vertical sliding osteotomy of the ramus, with or without bone graft: an improved technique, 1409

—. Chronic unilateral dislocation of the mandibular joint treated surgically by a high condylectomy, 12

COBB, JAMES R. Superiosteal Vitallium implants in dogs, 1153

COBE, HERBERT M. Investigations of a new dental chemotherapeutic agent in the presence of blood, 678

COHEN, LAWRENCE. Fractures of the maxillary tuberosity occurring during tooth extraction, 409

COHEN, M. M., JORESS, S. M., AND CALISTI, L. P. Bacteriologic study of infected deciduous molars, 1382

COOK, THOMAS J., AND MCKENZIE, JOHN S. The gingivae, 802

COPLAND, JAMES. Diagnosis of mandibular joint dysfunction, 1106

CRANDELL, C. E., AND TRUEBLOOD, S. NEAL. Roentgenographic findings in edentulous areas, 1343

CRANIN, A. NORMAN, AND CRANIN, SAMUEL L. Synergistic basal narcosedation, 176

CRANIN, SAMUEL L. (*See* CRANIN AND CRANIN), 176

CROUCH, STUART A. (*See* POYTON, MORGAN, AND CROUCH), 964

CROWLEY, MARY C. (*See* SCHMIDT, CROWLEY, HARNER, AND BURKMAN), 80

D

DARLINGTON, CHARLES G. (*See* GRUPP, PROTZEL, AND DARLINGTON), 308

DEMP, SHELIAH. (*See* GROSSMAN AND DEMP), 1130

DIRLAM, JAMES H. (*See* LINENBERG, DIRLAM, AND HARPOLE), 59

DORE, GEORGE D., JR. Tetracycline with glucosamine (Cosa-Tetracyn) in oral surgery, 530

DOUGLAS, MAX H. A new intravenous barbiturate for pediatric anesthesia in difficult and handicapped dental patients, 686

DRAUS, FRANK J., AND LEUNG, S. WAH. A bovine gland extract for calculus formation, 224

DRISCOLL, EDWARD J. (*See* GAMBLE AND DRISCOLL), 104

DUROCHER, ROY T. (*See* SHIP, MORRIS, DUROCHER, AND BURKET), 1191, 1317, 1438

DUTE, HAROLD L. (*See* MARSHALL, MASSLER, AND DUTE), 208

DUTZ, WERNER. (*See* KUTSCHER, FRATTA, ZEGARELLI, AND DUTZ), 937

E

ENGLAND, L. C. Ameloblastoma of the mandible, 648

EPSTEIN, IRWIN A. Classification in endodontics, 1258

ERICKSON, R. I., WAITE, D. E., AND WILKISON, R. H. A study of dry sockets, 1046

ESELMAN, J. C. Factors contributing to direct radiation reduction in dentistry, 432

F

FELDMAN, ARNOLD. (*See* RITCHEY, FELDMAN, AND GREER), 188

FIELD, HARRY J., AND ACKERMAN, ALFRED A. Osteosclerosis in endentulous mandible simulating carious unerupted cuspid, 668

FINDLAY, IAN A., AND NICHOLL, B. Tooth extraction in patients with hemophilia, 1167

FITZGERALD, GORDON M. A roentgenologic rebuttal, 1218

FITZGERALD, R. J., HAMPP, E. G., AND STANLEY, H. R. Studies on the survival of parenterally inoculated oral treponemes in the guinea pig, 883

FLEMING, HAROLD S. Parotid gland heterotransplants and Shope papilloma virus, 598

FONSECA, EDMUNDO P. (*See* VIANNA AND FONSECA), 33

FORSBERG, ARNE, LAGERGREN, CURT, AND LÖNNERBLAD, TORE. Dental calculus, 1051

FRATTA, ITALO. (*See* KUTSCHER, FRATTA, ZEGARELLI, AND DUTZ), 937

FREDRICKS, WALTER H. (*See* LARGE, NIEBEL, AND FREDRICKS), 1462

FREUND, H. ROBERT. (*See* SANDLER, STAHL, CAHN, AND FREUND), 994

FRY, ARTHUR E., WATKINS, ROBERT F., AND PHATAK, NILKANTH M. Topical use of corticosteroids for the relief of pain sensitivity of dentine and pulp, 594

G

GAMBLE, JACK W., AND DRISCOLL, EDWARD J. Oral manifestations of macroglobulinemia of Waldenström, 104

GARDNER, ALVIN F. (*See* AISENBERG, GARDNER, AND INMAN), 1356

GARDNER, J. A., AND HANFT, R. J. Chronic obstructive sialadenitis of the sublingual gland, 1295

— AND —. An ossifying fibroma of the mandible, 1288

GLENN, FRANCES B., AND STANLEY, HAROLD R., JR. Dilaceration of a mandibular permanent incisor, 1249

GOLDHABER, PAUL. (*See* ZACKIN AND GOLDHABER), 1267

GOLDMAN, HENRY M. Curettage by ultrasonic instrument, 43

GORDON, ROBERT B. (*See* QUINTARELLI, GORDON, AND CHAUNCEY), 875

GORES, ROBERT J. (*See* QUINLEY, ROYER, AND GORES), 38

—. (*See* STEG, GORES, THOMPSON, AND OWEN), 671

GORLIN, ROBERT J., AND CHAUDHRY, ANAND P. Unusual stomadromes—pachyonychia congenita, lipoid proteinosis, Reiter's syndrome, epidermolysis bullosa, Bonnevie-Ullrich-Turner's syndrome, and multiple hereditary telangiectasia, 257

—, —, AND KELLN, ELMER E. Oral manifestations of the Fitzgerald-Gardner, Pringle-Bourneville, Robin, adrenogenital, and Hurler-Pfaundler syndromes, 1233

—. (*See* CHAUDHRY, BURKE, AND GORLIN), 584

—. (*See* CHAUDHRY, BURKE, STOESZ, AND GORLIN), 716

—. (*See* CHAUDHRY AND GORLIN), 449

—. (*See* CHAUDHRY, GORLIN, AND MITCHELL), 452

—. (*See* CHAUDHRY, GORLIN, AND MOSSER), 269

—. (*See* CHAUDHRY, GORLIN, AND REYNOLDS), 578

—. (*See* KELLN, CHAUDHRY, AND GORLIN), 1245

GRANT, THEODORE S. (*See* POULTON AND GRANT), 1089

GREEN, DAVID. Stereomicroscopic study of 700 root apices of maxillary and mandibular posterior teeth, 728

GREER, WILLIAM. (*See* RITCHEY, FELDMAN, AND GREER), 188

GRIMM, DAVID H. (*See* WALKER, GRIMM, AND WAY), 1214

GROSSMAN, LOUIS I., AND DEMP, SHELIAH. Evaluation of a method to determine the presence of a focus of infection, 1130

GRUPP, SEYMOUR, PROTZEL, MARTIN S., AND DARLINGTON, CHARLES G. The fate of the human follicular sac in heterologous transplantation, 308

GUPTA, OM P., BLECHMAN, HARRY, AND STAHL, S. SIGMUND. The effects of desalivation on periodontal tissues of the Syrian hamster, 470

GYARMATI, I. Oral change in Sturge-Weber's disease, 795

H

HAMPP, E. G. (*See* FITZGERALD, HAMPP, AND STANLEY), 883

HANFT, R. J. (*See* GARDNER AND HANFT), 1288, 1295

HANRATTY, WILLIAM J. Hodgkin's disease treated with nitrogen mustard and radiation, complicated by osteomyelitis of the mandible, 651

HARNER, VERNA. (*See* SCHMIDT, CROWLEY, HARNER, AND BURKMAN), 80

HARPOLE, H. JAMES. (*See* LINENBERG, DIRLAM, AND HARPOLE), 59

—. (*See* SCHMITT, INGRAM, AND HARPOLE), 791

HAYES, RAYMOND L. Idiopathic internal resorption of teeth, 723

HELSHAM, R. W., AND BUCHANAN, G. Keratoacanthoma of the oral cavity, 844

HERLANDS, ROBERT E. (*See* ZEGARELLI, KUTSCHER, SILVERS, BEUBE, STERN, BERMAN, AND HERLANDS), 170

HIATT, W. H. Choice of anti-inflammatory agents in root resection, 396

HINIKER, J. J. (*See* UMIKER, LAMPE, RAPP, AND HINIKER), 897

HOFFER, OSCAR, AND VOGEL, GIORGIO. Mixed tumor of the salivary glands of the mandible, 519

HOLTON, PHILIP J. Histologic changes produced by pressure injection of whole blood into the intermedullary spaces of dog mandibles, 605

HUGHES, N. C. (*See* WHITLOCK AND HUGHES), 23

HURT, WILLIAM C. Periadenitis mucosa necrotica recurrens, 750

HUSSER, HORACE F. (*See* WINTERS, HUSSER, SIMARD, AND SHEBL), 76

HYSON, JOHN M., JR. Use of buccal streptokinase in the treatment of infection and edema, 812

I

INGLE, JOHN I. Alveolar osteoporosis and pulpal death associated with compulsive bruxism, 1371

INGRAM, R. C. (*See* SCHMITT, INGRAM, AND HARPOLE), 791

INMAN, B. WALLACE. (*See* AISENBERG AND INMAN), 1352

INMAN, C. L., SR. (*See* AISENBERG, GARDNER, AND INMAN), 1356

J

JORESS, S. M. (*See* COHEN, JORESS, AND CALISTI), 1382

K

KAPLOWITZ, BERNARD. (*See* ROSSMAN, KAPLOWITZ, AND BALDINGER), 361

KAPSIMALIS, PETER. (*See* PARRIS AND KAPSIMALIS), 982

KELLN, ELMER E., CHAUDHRY, ANAND P., AND GORLIN, ROBERT J. Oral manifestations of Crouzon's disease, 1245

—. (*See* GORLIN, CHAUDHRY, AND KELLN), 1233

KOHLER, CHARLES A., AND RAMFJORD, SIGURD P. Healing of gingival mucoperiosteal flaps, 89

KOWALSKI, KASIMER. Endotracheal anesthesia and trauma incident to endotracheal intubation, 543

KOZAM, GEORGE, AND BURNETT, GEORGE W. The effect of local anesthetics on the respiration of dental pulp, 229

KOZLOV, MARVIN, AND MASSLER, MAURY. Histologic effects of various drugs on amputated pulps of rat molars, 455

KRESHOVER, SEYMOUR J. Prenatal factors in oral pathologic conditions, 569

KUTSCHER, AUSTIN H., FRATTA, ITALO, ZEGARELLI, EDWARD V., AND DUTZ, WERNER. Triamcinolone acetonide, 937

—, ZEGARELLI, EDWARD V., AND PEARSON, HENRY. A color shade chart for study of the oral mucous membranes: preparation and construction, 1061

—. (*See* CAMPBELL, SEGUIN, KUTSCHER, AND ZEGARELLI), 1273

—. (*See* ZEGARELLI, KUTSCHER, SILVERS, BEUBE, STERN, BERMAN, AND HERLANDS), 170

L

LAGERGREN, CURT. (*See* FORSBERG, LAGERGREN, AND LÖNNERBLAD), 1051

LAMPE, I. (*See* UMIKER, LAMPE, RAPP, AND HINIKER), 897

LARGE, NELSON D., NIEBEL, HAROLD H., AND FREDRICKS, WALTER H. Myxoma of the jaws, 1462

LEUNG, S. WAH. (*See* DRAUS AND LEUNG), 224

LEVIN, LAWRENCE. (*See* MARK AND LEVIN), 401

LIBERATORE, THEODORE R., AND SAZIMA, HENRY J. Fibromyxoma and accompanying episodic symptoms, 142

LINENBERG, WILLIAM B., DIRLAM, JAMES H., AND HARPOLE, H. JAMES. Doriden: A quieting agent for anxiety in oral surgery, 59

LÖNNERBLAD, TORE. (*See* FORSBERG, LAGERGREN, AND LÖNNERBLAD), 1051

LOVESTEDT, STANLEY A. (*See* STAFNE AND LOVESTEDT), 1445

LOVETT, DUANE W. Roentgenographic follow-up of surgically introduced intramedullary pin in the mandible, 1229

Mc

MCCARTHY, FRANCIS P., MCCARTHY, PHILIP L., AND SHKLAR, GERALD. Chronic desquamative gingivitis: a reconsideration, 1300

MCCARTHY, PHILIP L. (*See* MCCARTHY, MCCARTHY, AND SHKLAR), 1300

MCCRAY, RICHARD E., JR. Primary amyloidosis of the tongue, 534

MCKELVEY, LOWELL E., ALBRIGHT, CECIL R., AND PRAZAK, GEORGE. Multiple hereditary familial epithelial cysts of the jaws with the associated anomaly of trichoepithelioma, 111

MCKENZIE, JOHN S. (*See* COOK AND MCKENZIE), 802

M

MADAN, RAFAEL. Ameloblastoma developing from a dentigerous cyst, 781

MAISTO, O. A. (*See* CABRINI, MAISTO, AND MANFREDI), 868

MANFREDI, E. E. (*See* CABRINI, MAISTO, AND MANFREDI), 868

MANSON-HING, L. R. Use of dental x-rays in roentgenography of the palatopharyngeal mechanism, 1085

— AND CHERASKIN, E. Relationship of three-hour blood glucose to oral roentgenographic findings, 1447

—. (*See* CHERASKIN AND MANSON-HING), 819

MARK, HOWARD I., AND LEVIN, LAWRENCE. Dentigerous cyst involving a supernumerary maxillary molar with encroachment on the maxillary antrum, 401

MARSHALL, F. JAMES. Root fracture, 1485

—, MASSLER, MAURY, AND DUTE, HAROLD L. Effects of endodontic treatments on permeability of root dentine, 208

MASHBERG, ARTHUR, THOMA, KURT H., AND WASILEWSKI, EDWARD J. Olfactory neuroblastoma (esthesioneuroepithelioma) of the maxillary sinus, 908

MASSLER, MAURY. (*See* KOZLOV AND MASSLER), 455

—. (*See* MARSHALL, MASSLER, AND DUTE), 208

MEYER, IRVING, AND SHKLAR, GERALD. Multiple malignant tumors involving the oral mucosa and the gastrointestinal tract, 295

MITCHELL, DAVID F., AND TARPLEE, ROBERT E. Painful pulpitis, 1360

—. (*See* CHAUDHRY, GORLIN, AND MITCHELL), 452

MONICA, WOODROW S. Anaplasia, 581

MONOMACK, OLGA. (*See* WALKER, GRIMM, AND WAY), 1214

MOOS, WALTER S. (*See* YALE, MOOS, AND VIDEKA), 1081

MORGAN, ALBERT F., STEWART, FRANK W., AND STEIN, IGNATIUS J. Double lip, 236

MORGAN, GEORGE A., AND POYTON, H. GUY. Bilateral dens in dente, 63

—. (*See* POYTON, MORGAN, AND CROUCH), 964

MORRIS, ALVIN L. (*See* SHIP, MORRIS, DUROCHER, AND BURKET), 1191, 1317, 1438

MORRIS, ESTELL E. (*See* PING AND MORRIS), 539

MOSKOW, BERNARD S., AND TENEN, CHARLES. Familial intestinal polyposis with melanosis oris: the syndrome of Peutz and Jeghers, 924

MOSSER, DONN G. (*See* CHAUDHRY, GORLIN, AND MOSSER), 269

N

NAWFEL, ELIAS R. Cardiac arrest, 427

NEWTON, WALTER L. (*See* BAER AND NEWTON), 1134

NICHOLL, B. (*See* FINDLAY AND NICHOLL), 1167

NIEBEL, HAROLD H. (*See* LARGE, NIEBEL, AND FREDRICKS), 1462

O

OLIVER, STANTON J. Experimental use of proteolytic enzymes in the treatment of oral and associated lesions, 641

OSHEROFF, BORIS J. (*See* SHIP, WILLIAMS, AND OSHEROFF), 630

OWEN, CHARLES A., JR. (*See* STEG, GORES, THOMPSON, AND OWEN), 671

P

PARRIS, LEONARD, AND KAPSIMALIS, PETER. The effect of temperature change on the sealing properties of temporary filling materials, 982

PEARSON, HENRY. (*See* KUTSCHER, ZEGARELLI, AND PEARSON), 1061

PHATAK, NILKANTH M. (*See* FRY, WATKINS, AND PHATAK), 594

PING, RONALD S., AND MORRIS, ESTELL E. Ilosone, the propionyl ester of erythromycin, 539

PIRILÄ, VEIKKO, AND RANTANEN, AIMO V. Root canal treatment with bacitracin-neomycin as cause of flare-up of allergic eczema, 589

PIZER, MARVIN E. Cervicofacial actinomycosis of the mandible, 775

POOL, M. L. (*See* SPANGENBERG AND POOL), 552

POULTON, DONALD R., AND GRANT, THEODORE S. Development of a profile-intensifying shield for cephalometric roentgenography, 1089

POYTON, H. GUY, MORGAN, GEORGE A., AND CROUCH, STUART A. Recurring supernumerary mandibular premolars, 964

—. (*See* MORGAN AND POYTON), 63

PRAZAK, GEORGE. (*See* MCKELVEY, ALBRIGHT, AND PRAZAK), 111

PRIGMORE, JOHN R. (*See* SHANNON AND PRIGMORE), 878, 1013, 1488

—. (*See* SHANNON, PRIGMORE, AND BROOKS), 1010

PROTZEL, MARTIN S. (*See* GRUPP, PROTZEL, AND DARLINGTON), 308

PROVENZA, D. VINCENT, BIDDIX, JOSEPH C., AND CHENG, THOMAS C. Studies on the etiology of periodontosis. II. Glomera as vascular components in the periodontal membrane, 157

Q

QUART, A., STAHL, S. S., AND SORRIN, S. Gingival changes observed in arteriosclerotic men, 1181

QUINLEY, JAMES F., ROYER, R. QUENTIN, AND GORES, ROBERT J. "Dry socket" after mandibular odontectomy and use of soluble tetracycline hydrochloride, 38

QUINN, JAMES H. Congenital epidermoid cyst of anterior half of tongue, 1283

QUINTARELLI, GIULIANO, GORDON, ROBERT B., AND CHAUNCEY, HOWARD H. The effect of parotid gland extract on the hard and soft tissues of growing mice, 875

R

RAHIMO, AZIZ A., AND SHIMASAKI, WALTER W. Mercurial necrosis of the cheek, 54

RAMFJORD, SIGURD P. Recurrent herpetic gingivostomatitis treated with gamma globulin, 165

—. (*See* KOHLER AND RAMFJORD), 89

RANTANEN, AIMO V. (*See* PIRILÄ AND RANTANEN), 589

RAPP, R. (*See* UMIKER, LAMPE, RAPP, AND HINIKER), 897

RECANT, BENJAMIN S. Methohexital sodium, 1330

REYNOLDS, D. H. (*See* CHAUDHRY, GORLIN, AND REYNOLDS), 578

RICHARDS, ALBERT G. Dental x-ray equipment of the future, 194

—. Radiation protection via the pinhole camera, 953

RICKLES, NORMAN H., AND BERNIER, JOSEPH L. Cat-scratch disease, 282

RITCHEY, BERYL, FELDMAN, ARNOLD, AND GREER, WILLIAM. Roentgenography of enamel, 188

ROBINSON, ERIC R. (*See* ADILMAN AND ROBINSON), 918

ROBINSON, H. B. G. (*See* STANLEY, ZANDER, ROBINSON, AND BERNIER), 329

ROSSMAN, SAMUEL R., KAPLOWITZ, BERNARD, AND BALDINGER, S. ROBERT. Therapy of the endodontically and periodontally involved tooth, 361

ROYER, R. QUENTIN. (*See* QUINLEY, ROYER, AND GORES), 38

S

SALA, HERNANDO L., AND SPERA, JOSÉ NORBERTO. Resection of the upper jaw and its immediate restoration, 1033

SANDLER, HENRY C., STAHL, S. SIGMUND, CAHN, LESTER R., AND FREUND, H. ROBERT. Exfoliative cytology for detection of early mouth cancer, 994

SAZIMA, HENRY J. (*See* LIBERATORE AND SAZIMA), 142

SCHMIDT, HENRY S., CROWLEY, MARY C., HARNER, VERNA, AND BURKMAN, N. WEIR. A bacteriologic report of an investigation to study in vivo the effectiveness of certain drugs for the sterilization of carious dentine, 80

SCHMITT, JACK, INGRAM, R. C., AND HARPOLE, H. JAMES. Hematoma following dental extractions in a patient receiving unreported anticoagulant therapy, 791

SCHULTZ, LOUIS W., ATTERBURY, ROBERT A., AND VAZIRANI, SUNDER J. Odontogenic cyst of the mandible, 523

— AND VAZIRANI, SUNDER J. Odontoma of the maxilla, 513

— AND —. Plastic closure of oronasal fistula, 146

— AND —. Use of sclerosing solution in treatment of ameloblastoma, 150

—, —, AND BOLDEN, THEODORE E. Unilateral hyperplasia and exostosis of the mandibular condyle, 387

SCIGLIANO, JOHN. (*See* BAER, SUMNER, AND SCIGLIANO), 494

SEGUIN, LUCILE. (*See* CAMPBELL, SEGUIN, KUTSCHER, AND ZEGARELLI), 1273

SELTZER, SAMUEL. (*See* BENDER, SELTZER, AND YERMISH), 353

SHANNON, IRA L., AND BROOKS, ROBERT A. The effect of pilocarpine stimulation on the histologic appearance and the sodium and potassium content of rat submaxillo-sublingual glands, 240

— AND PRIGMORE, JOHN R. Parotid fluid as a medium for the determination of human adrenocortical status, 878

— AND —. Parotid fluid flow rate, 1488

— AND —. Parotid gland flow rate and parotid fluid urea concentration, 1013

—, —, AND BROOKS, ROBERT A. Glucose concentrations in parotid fluid and blood serum following intravenous glucose loading, 1010

SHARP, GEORGE S. The treatment of precancerous oral mucous membranes, 1065

SHEBL, JOSEPH J. (*See* WINTERS, HUSSER, SIMARD, AND SHEBL), 76

SHEINGORN, ABE. (*See* BARILE AND SHEINGORN), 756

SHIMASAKI, WALTER W. (*See* RAHIMO AND SHIMASAKI), 54

SHIP, IRWIN I. The response of systolic and diastolic blood pressures to dental stress, 499

—, MORRIS, ALVIN L., DUROCHER, ROY T., AND BURKET, LESTER W. Recurrent aphthous ulcerations and recurrent herpes labialis in a professional school student population. I. Experience, 1191

—, —, —, AND —. Recurrent aphthous ulcerations and recurrent herpes labialis in a professional school student population. II. Medical history, 1317

—, —, —, AND —. Recurrent aphthous ulcerations and recurrent herpes labialis in a professional school student population. III. Oral examinations, 1438

— AND WHITE, CARL L. Physiologic response to dental stress, 368

—, WILLIAMS, ANDERSON F., AND OSHEROFF, BORIS J. Development and clinical investigation of a new oral surface anesthetic for acute and chronic oral lesions, 630

SHKLAR, GERALD. (*See* MCCARTHY, MCCARTHY, AND SHKLAR), 1300

—. (*See* MEYER AND SHKLAR), 295

SHORE, NATHAN ALLEN. The interpretation of temporomandibular joint roentgenograms, 341

SIBLEY, LONNIE C. Management of root fracture, 1475

SICHER, HARRY. (*See* CHOUKAS AND SICHER), 1203

SILHA, ROBERT E. Horizontal angulation in maxillary cuspid roentgenography, 710

SILVERMAN, SOL, JR., AND WARE, WILLIAM H. Comparisons of histologic, cytologic, and clinical findings in intraoral leukoplakia and associated carcinoma, 412

SILVERS, HERBERT F. (*See* ZEGARELLI, KUTSCHER, SILVERS, BEUBE, STERN, BERMAN, AND HERLANDS), 170

SIMARD, ERNEST E. (*See* WINTERS, HUSSER, SIMARD, AND SHEBL), 76

SIMON, G. T. (*See* TOPAZIAN AND SIMON), 1038

SMALL, GILBERT S. (*See* SMALL AND SMALL), 320

SMALL, IRWIN A., AND SMALL, GILBERT S. Infantile hemangioendothelioma of the tongue, 320

SMITH, JAMES F. Ameloblastoma, 1253

—. Bronchiectasis and chronic lung abscess from aspiration of a dental bridge, 1023

—. Malignant melanoma primary in the oral cavity, 971

—. An oral benign lesion progressing to epidermoid carcinoma with metastasis to the lung, 977

—. Peripheral giant-cell granuloma, 974

—. Recurrent adenoid cystic carcinoma of the submaxillary gland, 1103

—. The relationship of oral cancer to generalized connective tissue changes, 838

—. Tumors of the salivary glands, with a review of 150 cases, 850

SORRIN, S. (*See* QUART, STAHL, AND SORRIN), 1181

SPANGENBERG, HARRY D., JR., AND POOL, M. L. A 65 or a 90 kilovolt x-ray machine? 552

SPERA, JOSÉ NORBERTO. (*See* SALA AND SPERA), 1033

SPITZER, RICHARD. Partial ankyloglossia, 787

STAFNE, EDWARD C., AND LOVESTEDT, STANLEY A. Osteoporosis of the jaws associated with hypercortisonism, 1445

STAHL, S. S. Response of the periodontium, pulp, and salivary glands to gingival and tooth injury in young adult male rats. I. Periodontal tissues, 613

—. Response of the periodontium, pulp, and salivary glands to gingival and tooth injury in young adult male rats. II. Pulp and periapical tissues, 734

—. Response of the periodontium, pulp, and salivary glands to gingival and tooth injury in young adult male rats. III. Submaxillary glands, 870

—. (*See* GUPTA, BLECHMAN, AND STAHL), 470

—. (*See* QUART, STAHL, AND SORRIN), 1181

—. (*See* SANDLER, STAHL, CAHN, AND FREUND), 994

STANLEY, HAROLD R., ZANDER, HELMUT, ROBINSON, H. B. G., AND BERNIER, JOSEPH L. Symposium on pulp reaction to operative procedures, 329

—. (*See* FITZGERALD, HAMPP, AND STANLEY), 883

—. (*See* GLENN AND STANLEY), 1249

STANMEYER, WILLIAM R. (*See* ADAMS AND STANMEYER), 117

STEG, RONALD F., GORES, ROBERT J., THOMPSON, JOHN H., JR., AND OWEN, CHARLES A., JR. Bleeding due to deficiency of plasma thromboplastin antecedent (PTA) and plasma thromboplastin component (PTC), 671

STEIN, IGNATIUS J. (*See* MORGAN, STEWART, AND STEIN), 236

STERN, IRVING B. (*See* ZEGARELLI, KUTSCHER, SILVERS, BEUBE, STERN, BERMAN, AND HERLANDS), 170

STEWART, FRANK W. (*See* MORGAN, STEWART, AND STEIN), 236

STOCKDALE, C. R. Branchial carcinoma, 136

STOESZ, A. R. (*See* CHAUDHRY, BURKE, STOESZ, AND GORLIN), 716

SUMNER, CHARLES F., III. (*See* BAER, SUMNER, AND SCIGLIANO), 494

SWANSON, ALVA E., AND WHITE, JAMES B. The unusual occurrence of a large sublingual neurilemmoma, 1163

SWERDLOFF, GEORGE. Double lip, 627

T

TARPLEE, ROBERT E. (*See* MITCHELL AND TARPLEE), 1360

TENEN, CHARLES. (*See* MOSKOW AND TENEN), 924

TERNER, CORNELIA. Retention of a lower first permanent molar, 1425

THILANDER, H. (*See* BERGENHOLTZ AND THILANDER), 767

THOMA, KURT H. Progressive atrophy of the mandible complicated by fractures: its reconstruction, 4

—. (*See* MASHBERG, THOMA, AND WASILEWSKI), 908

THOMPSON, JOHN H., JR. (*See* STEG, GORES, THOMPSON, AND OWEN), 671

TOPAZIAN, RICHARD G., AND SIMON, G. T. Adenoameloblastoma, 1038

TRUEBLOOD, S. NEAL. (*See* CRANDELL AND TRUEBLOOD), 1343

U

UMIKER, W. O., LAMPE, I., RAPP, R., AND HINIKER, J. J. Oral smears in the diagnosis of carcinoma and premalignant lesions, 897

V

VAZIRANI, SUNDER J. (*See* SCHULTZ, ATTERBURY, AND VAZIRANI), 523

—. (*See* SCHULTZ AND VAZIRANI), 146, 150, 513

—. (*See* SCHULTZ, VAZIRANI, AND BOLDEN), 387

VERNALE, CHARLES A. Cardiovascular responses to local dental anesthesia with epinephrine in normotensive and hypertensive subjects, 942

VIANNA, CICERO B., AND FONSECA, EDMUNDO P. The use of a temporary maxillofacial prosthesis in surgical-oncologic cases, 33

VIDEKA, MICHAEL A. (*See* YALE, MOOS, AND VIDEKA), 1081

VILLA, VICTORINO G., AND BUNAG, CELSO A. Neurogenic fibrosarcoma in the mandible observed about one month after extraction of a lower third molar, 203

VOGEL, GIORGIO. (*See* HOFFER AND VOGEL), 519

W

WAGGENER, DONALD T. Roentgenographic localization of unerupted teeth, 439

WAITE, D. E. (*See* ERICKSON, WAITE, AND WILKISON), 1046

WALKER, DONALD E., GRIMM, DAVID H., AND WAY, E. LEONG (WITH THE ASSISTANCE OF MONOMACK, OLGA). An evaluation of the analgesic properties of a salicylamide derivative in comparison with a placebo and aspirin, 1214

WARE, WILLIAM H. (*See* SILVERMAN AND WARE), 412

WASILEWSKI, EDWARD J. (*See* MASHBERG, THOMA, AND WASILEWSKI), 908

WATKINS, ROBERT F. (*See* FRY, WATKINS, AND PHATAK), 594

WAY, E. LEONG. (*See* WALKER, GRIMM, AND WAY), 1214

WEINREB, MAX M. The conservative treatment of extensive areas of periapical pathology, 858

WEINSTOCK, SIDNEY S. Methohexital sodium, anesthesia for the ambulant dental patient, 1072

WHITE, CARL L. (*See* SHIP AND WHITE), 368

WHITE, JAMES B. (*See* SWANSON AND WHITE), 1163

WHITLOCK, R. I. H., AND HUGHES, N. C. Solitary myeloma of mandible, 23

WILKISON, R. H. (*See* ERICKSON, WAITE, AND WILKISON), 1046

WILLIAMS, ANDERSON F. (*See* SHIP, WILLIAMS, AND OSHEROFF), 630

WINTERS, STANLEY E., HUSSER, HORACE F., SIMARD, ERNEST E., AND SHEBL, JOSEPH J. Neurofibromatosis (Von Recklinghausen's disease) with involvement of the mandible, 76

Y

YALE, SEYMOUR H., MOOS, WALTER S., AND VIDEKA, MICHAEL A. Measurement of gonadal dose in children during intraoral radiography, 1081

YERMISH, MORRIS. (*See* BENDER, SELTZER, AND YERMISH), 353

Z

ZACKIN, S. JEROME, AND GOLDHABER, PAUL. Experimental production of oral ulcerations in the rat, 1267

ZANDER, HELMUT. (*See* STANLEY, ZANDER, ROBINSON, AND BERNIER), 329

ZEGARELLI, EDWARD V., KUTSCHER, AUSTIN H., SILVERS, HERBERT F., BEUBE, FRANK E., STERN, IRVING B., BERMAN, CHARLES L., AND HERLANDS, ROBERT E. Triamcinolone acetonide in the treatment of acute and chronic lesions of the oral mucous membranes, 170

—. (*See* CAMPBELL, SEGUIN, KUTSCHER, AND ZEGARELLI), 1273

—. (*See* KUTSCHER, FRATTA, ZEGARELLI, AND DUTZ), 937

—. (*See* KUTSCHER, ZEGARELLI, AND PEARSON), 1061

SUBJECT INDEX*

A

Abscess, chronic lung, and bronchiectasis from aspiration of dental bridge, 1023
Abstracts of current literature, 127, 251, 382, 639, 894, 1022, 1149, 1407, 1512
Actinomycetes, oral, 384 (Abst.)
Actinomycosis, cervicofacial, of mandible, 775
Adenoameloblastoma, 383 (Abst.), 1038
Adenoid cystic carcinoma, recurrent, of submaxillary gland, 1103
Adrenocortical status, human, parotid fluid as medium for determining, 878
Adrenogenital, Fitzgerald-Gardner, Pringle-Bourneville, Robin, and Hurler-Pfaundler syndromes, oral manifestations, 1233
Allergic eczema, root canal treatment with bacitracin-neomycin as cause of flare-up, 589
Allergy and dental medicine, 1512 (B. rev.)
Alpha Omega, Greater Miami chapter, 1276, 1406
Alveolar bone loss; relationship of three-hour blood glucose to oral roentgenographic findings, 1447
  osteoporosis and pulpal death associated with compulsive bruxism, 1371
Ameloblastoma, 1253
  arising within globulomaxillary cyst, 1352
  developing from dentigerous cyst, 781
  of mandible, 648
  use of sclerosing solution in treatment, 150
American Academy of Dental Medicine announces 1961 cruise convention, 1506
American Academy of Maxillofacial Prosthetics, 1147
American Academy of Oral Pathology, 121, 761
  proceedings, 257
American Academy of Periodontology, 761
American Association of Endodontists, 891, 1019, 1400, 1509
American Board of Oral Pathology, 761
American Dental Society of Anesthesiology, Illinois section, 1506
American Institute of Oral Biology, 761
  case history service, 1400
American men of science, 382 (B. rev.)
American Society of Dentistry for Children, 1019
American Society of Psychosomatic Dentistry and Medicine, Inc., 246
Amino-acid ointment, antibiotic-induced rectal itch treated with, 896 (Abst.)
Amphotericin B, activity against *Candida albicans*, 1273
Amyloidosis, primary, of tongue, 534
Analgesic properties of salicylamide derivative in comparison with placebo and aspirin, 1214
Anaplasia, 581
Anatomy (*See* Oral Anatomy, Surgical Anatomy)
Anesthesia, endotracheal, and trauma incident to endotracheal intubation, 543
  in general practice, 252 (Abst.)
  induction in young children, 1512 (Abst.)
  local dental, with epinephrine, cardiovascular responses in normotensive and hypertensive subjects, 942
  methohexital sodium, for ambulant dental patient, 1072
  pediatric, in difficult and handicapped patients, new intravenous barbiturate, 686
Anesthesiology, 176, 427, 543, 686, 942, 1072, 1330
  hypnosis as adjunct, 1513 (Abst.)
Anesthetic(s), local, effect on respiration of dental pulp, 229
  new nonexplosive volatile: Fluothane, 640 (Abst.)
  oral surface, for acute and chronic oral lesions, 630
Ankyloglossia, partial, 787
Announcements, 121, 246, 377, 508, 637, 761, 891, 1019, 1145, 1276, 1400, 1506
Antarctic isolation, prolonged, effects on oral and intestinal bacteria, 117
Antibiotic-induced rectal itch treated with amino-acid ointment, 896 (Abst.)
Anticoagulant therapy, unreported, hematoma following dental extractions in patient receiving, 791
Anti-inflammatory agents in root resection, choice of, 396
Antrum, carcinoma of, 269
  maxillary, dentigerous cyst involving supernumerary maxillary molar with encroachment on, 401
Anxiety in oral surgery, quieting agent for, 59

*January, pp. 1-128; February, pp. 129-254; March, pp. 255-384; April, pp. 385-512; May, pp. 513-640; June, pp. 641-766; July, pp. 767-896; August, pp. 897-1022; September, pp. 1023-1150; October, pp. 1151-1280; November, pp. 1281-1408; December, pp. 1409-1538.

Apatite as phantom material in roentgenography of enamel, 188
Aphthous lesions; periadenitis mucosa necrotica recurrens, 750
ulcerations, recurrent, and recurrent herpes labialis in professional school student population, 1191, 1317, 1438
Armed Forces Institute of Pathology, 121, 1506
new chief at Dental and Oral Pathology Division, 1460
Armed Forces and Public Health Service, section by, 104, 236, 368, 494, 627, 750, 878, 1010, 1134, 1387, 1488
Army Graduate School of Dentistry, 892, 1021
Arteriosclerotic men, gingival changes observed in, 1181
Artifact, roentgenographic, 1458 (Roentgeno-Oddities)
Atrophy, progressive, of mandible, complicated by fractures, 4
Australian Dental Congress, sixteenth, 892

B

Bacitracin-neomycin, root canal treatment with, as cause of flare-up of allergic eczema, 589
Bacteremia, incidence in endodontic manipulation, 353
Bacteria, oral and intestinal, effects of prolonged Antarctic isolation on, 117
Bacterial invasion of blood after oral surgical procedures, 1022 (Abst.)
Bacteriology, 1273
Barbiturate anesthetic, new intravenous: methohexital sodium, 1330
new intravenous, for pediatric anesthesia in difficult and handicapped patients, 686
Basal-cell nevus, 252 (Abst.)
Benign neoplastic fibro-osseous lesion of mandible, 716
oral lesion progressing to epidermoid carcinoma with metastasis to lung, 977
Berkshire Conference in Periodontology and Oral Pathology, eleventh, 379
Bismuth stomatitis, 802
Black hairy tongue, biocenologic approach with new concepts of treatment, 384 (Abst.)
Bleeding due to deficiency of plasma thromboplastin antecedent and plasma thromboplastin component, 671
hypochlorite for control of, 1026
Blood, bacterial invasion after oral surgical procedures, 1022 (Abst.)
dyscrasias, gingival manifestations, 810
glucose normality, development by analysis of oral roentgenographic findings; normal glucose-tolerance pattern, 819
three-hour, relationship to oral roentgenographic findings, 1447
Blood—Cont'd
investigations of new dental chemotherapeutic agent in presence of, 678
pressures, systolic and diastolic, response to dental stress, 499
serum and parotid fluid, glucose concentrations following intravenous glucose loading, 1010
whole, histologic changes produced by pressure injections into intermedullary spaces of dog mandibles, 605
Bone(s), fractured and diseased, use of polyurethane polymer (Ostamer) in, 896 (Abst.)
loss due to osteomyelitis in line of fracture, reconstruction of mandible following, 663
Bonnevie-Ullrich-Turner's syndrome; unusual stomadromes, 257
Book reviews, 124, 382, 511, 764, 894, 1022, 1280, 1407, 1510
Books received, 251, 765
Boston University, 122, 247, 248, 380, 509, 637, 763, 892, 893, 1019, 1021, 1276, 1402
Bovine gland extract for calculus formation, 224
Branchial carcinoma, 136
Breast carcinoma metastatic to maxilla, 1099
Bronchiectasis and chronic lung abscess from aspiration of dental bridge, 1023
Bruxism as cause of headache, 639 (Abst.)
compulsive, alveolar osteoporosis and pulpal death associated with, 1371
Buccal streptokinase in treatment of infection and edema, 812

C

Calculus, dental, 1051
formation, bovine gland extract for, 224
Camera, pinhole, radiation protection via, 953
Cancer chemotherapy, 1149 (Abst.)
coordinators, 121
experimental production with tobacco tar and heat, 1513 (Abst.)
head and neck, exfoliative cytology applied to detection and treatment, 251 (Abst.)
intraoral, elective neck dissection for, 1150 (Abst.)
of mouth, early, exfoliative cytology for detection, 994
of oral cavity, early, with special reference to cancer of tongue, 895 (Abst.)
oral, exfoliative cytology in radiotherapy, 1512 (Abst.)
failures in treatment, 1408 (Abst.)
relationship to generalized connective tissue changes, 838
of tongue, early cancer of oral cavity with special reference to, 895 (Abst.)
Candida albicans, activity of amphotericin B against, 1273

Carcinoma of antrum, 269
associated with intraoral leukoplakia, comparisons of histologic, cytologic, and clinical findings, 412
branchial, 136
breast, metastatic to maxilla, 1099
epidermoid oral benign lesion progressing to, with metastasis to lung, 977
of gingival mucosa, 809
intraoral, end results and causes of failure in treatment, 1408 (Abst.)
radical neck dissection for cervical lymph node metastasis, 1514 (Abst.)
and premalignant lesions, oral smears in diagnosis, 897
recurrent adenoid cystic, of submaxillary gland, 1103
squamous-cell, of floor of mouth, treatment, 253 (Abst.)
of mandibular gingiva, effects of treatment, 252 (Abst.)
of tongue, treatment, 254 (Abst.)
Cardiac arrest, 427
Cardiovascular responses to local dental anesthesia with epinephrine in normotensive and hypertensive subjects, 942
Carious dentine, effectiveness of certain drugs for sterilization of, 80
unerupted cuspid, osteosclerosis in edentulous mandible simulating, 668
Cat-scratch disease, 283
Cephalometric roentgenography, development of profile-intensifying shield for, 1089
Cervicofacial actinomycosis of mandible, 775
Cheek, mercurial necrosis of, 54
Chemotherapeutic agent, new dental, investigations in presence of blood, 678
Chemotherapy of cancer, 1149 (Abst.)
Children, measurement of gonadal dose during intraoral radiography, 1081
preanesthetic medication for, 127 (Abst.)
young, induction of anesthesia in, 1512 (Abst.)
Classification in endodontics, 1258
College of Physicians and Surgeons of San Francisco, 763
Color shade chart for study of oral mucous membranes, 1061
Condyle, mandibular, exostosis of, and unilateral hyperplasia, 387
Condylectomy, high, chronic unilateral dislocation of mandibular joint treated surgically by, 12
Corticosteroid(s), new synthetic; triamcinolone acetonide, 937
topical use for relief of pain sensitivity of dentine and pulp, 594
Cosa-Tetracyn (tetracycline with glucosamine) in oral surgery, 530
Cranial injuries; management of upper facial and anterior cranial injuries, 383 (Abst.)
Crouzon's disease, oral manifestations, 1245
Cuspid, carious unerupted, osteosclerosis in edentulous mandible simulating, 668
Cyst(s), congenital epidermoid, of anterior half of tongue, 1283
dentigerous, ameloblastoma developing from, 781
involving supernumerary maxillary molar with encroachment on maxillary antrum, 401
globulomaxillary, ameloblastoma arising within, 1352
multiple hereditary familial epithelial, of jaws with associated anomaly of trichoepithelioma, 111
odontogenic, of mandible, 523
Cystadenoma, papillary, of minor salivary gland origin, 452
Cytochrome oxidase in normal and inflamed gingiva, histochemical study, 1501
Cytology, exfoliative, applied to detection and treatment of head and neck cancer, 251 (Abst.)
for detection of early mouth cancer, 994
in radiotherapy of oral cancer, 1512 (Abst.)
oral smears in diagnosis of carcinoma and premalignant lesions, 897

D

Dens in dente, bilateral, 63
multiple, 351 (Roentgeno-Oddities)
Dental bridge, aspiration of, resulting in bronchiectasis and chronic lung abscess, 1023
calculus, 1051
chemotherapeutic agent, investigations in presence of blood, 678
extractions, hematoma following, in patient receiving unreported anticoagulant therapy, 791
Guidance Council for Cerebral Palsy, 1400
medicine and allergy, 1512 (B. rev.)
offices, radiation dosage in, 696
origin of headache, 1314
patient, ambulant, methohexital sodium anesthesia for, 1072
practice administration, 1511 (B. rev.)
pulp, human, isolation and cultivation of filterable form resembling pleuropneumonia-like organisms from, 756
respiration, effect of local anesthetics, 229
roentgenology, 125 (B. rev.)
sepsis and intrathoracic suppuration, 895 (Abst.)
stress, physiologic response, 368
response of systolic and diastolic blood pressures, 499
tissues, mineralized, 70
x-ray equipment of future, 194
x-rays, use in roentgenography of palatopharyngeal mechanism, 1085
Dentigerous cyst, ameloblastoma developing from, 781
involving supernumerary maxillary molar with encroachment on maxillary antrum, 401

Dentine, carious, effectiveness of certain drugs for sterilization of, 80
incremental pattern in case of osteogenesis imperfecta, 70
and pulp, pain sensitivity of, topical use of corticosteroids for relief, 594
root, permeability of, effects of endodontic treatments, 208
Dentistry, factors contributing to direct radiation reduction in, 432
oral pathologist looks at practice of, 331
treatment planning in practice of, 126 (B. rev.)
Denture(s), complete, 1407 (B. rev.)
implant: indications and procedures, 125 (B. rev.)
prosthesis, partial removable, essentials, 126 (B. rev.)
Desalivation, effects on periodontal tissues of Syrian hamster, 470
Desquamative gingivitis, chronic, 1300
Diabetes, gingival manifestations, 808
Diagnosis of mandibular joint dysfunction, 1106
in oral surgery and oral medicine, studies in, 482, 994
oral, of systemic disease, 896 (Abst.)
Dilaceration of mandibular permanent incisor, 1249
Dilantin hypertrophy of gums, 804
Dislocation of mandibular joint, chronic unilateral, treated surgically by high condylectomy, 12
District of Columbia Dental Society, twenty-eighth annual postgraduate clinic, 246
Doriden: quieting agent for anxiety in oral surgery, 59
Double lip, 236, 627
Drugs, histologic effects on amputated pulps of rat molars, 455
for sterilization of carious dentine, 80
Dry socket(s), 1046
after mandibular odontectomy and use of soluble tetracycline hydrochloride, 38
Dysplasia, familial white folded, of mucous membranes, 252 (Abst.)

E

Eczema, allergic, root canal treatment with bacitracin-neomycin as cause of flare-up, 589
Edema and infection, buccal streptokinase in treatment, 812
Edentulous areas, roentgenographic findings, 1343
mandible, osteosclerosis simulating carious unerupted cuspid, 668
mouth, delayed treatment of unerupted teeth in, 918
Einstein, Albert, College of Medicine, Yeshiva University, 1279, 1406
Medical Center, 249, 380, 509
Emotionally disturbed and mentally retarded patients, dental use of meprobamate and promazine hydrochloride for, 423
Enamel, roentgenography, 188
Endodontic(s), 80, 208, 353, 455, 589, 723, 858, 982, 1130, 1258, 1371, 1475
classification in, 1258
manipulation, incidence of bacteremia in, 353
practice, 764 (B. rev.)
treatments, effects on permeability of root dentine, 208
Endodontically and periodontally involved tooth, therapy, 361
Endotracheal anesthesia and trauma incident to endotracheal intubation, 543
Enzymes, proteolytic, experimental use in treatment of oral and associated lesions, 641
Epidermoid carcinoma, oral benign lesion progressing to, with metastasis to lung, 977
cyst, congenital, of anterior half of tongue, 1283
Epidermolysis bullosa; unusual stomadromes, 257
Epinephrine, local dental anesthesia with, cardiovascular responses in normotensive and hypertensive subjects, 942
Epithelial cysts of jaws, multiple hereditary familial, with associated anomaly of trichoepithelioma, 111
Epulis, giant-cell, hyperparathyroidism first suspected through, 482
Errors, technical, 714, 968 (Roentgeno-Oddities)
Erythromycin, propionyl ester of (Ilosone), 539
Esthesioneuroepithelioma (olfactory neuroblastoma) of maxillary sinus, 908
Estrogenic hormone for treatment of leukoplakia buccalis and related lesions, 1513 (Abst.)
European Orthodontic Society, 1401
Exfoliative cytology applied to detection and treatment of head and neck cancer, 251 (Abst.)
for detection of early mouth cancer, 994
Exodontia, 38, 409, 668, 791, 918, 1046, 1167, 1425
Exostosis of mandibular condyle and unilateral hyperplasia, 387
Extraction(s), dental, hematoma following, in patient receiving unreported anticoagulant therapy, 791
of lower third molar, neurogenic fibrosarcoma in mandible observed about one month after, 203
tooth, fractures of maxillary tuberosity occurring during, 409
in patients with hemophilia, 1167

F

Facial injuries; management of upper facial and anterior cranial injuries, 383 (Abst.)
surgical treatment, 764 (B. rev.)
pain, 1512 (Abst.)
Familial internal resorption, 201 (Roentgeno-Oddities)
intestinal polyposis with melanosis oris: syndrome of Peutz and Jeghers, 924
white folded dysplasia of mucous membranes, 252 (Abst.)
Fauchard, Pierre, bicentennial of death of, 1506
Fibroma, ossifying, of mandible, 1288
Fibromyxoma and accompanying episodic symptoms, 142
Fibro-osseous lesion, benign neoplastic, of mandible, 716
Fibrosarcoma, neurogenic, in mandible observed about one month after extraction of lower third molar, 203
Filling materials, temporary, effect of temperature change on sealing properties, 982
Film reticulation, 567 (Roentgeno-Oddities)
First District Dental Society, 246, 377, 1145, 1276, 1406
Fistula, oronasal, plastic closure, 146
of Stensen's duct, correction, 382 (Abst.)
Fitzgerald-Gardner, Pringle-Bourneville, Robin, adrenogenital, and Hurler-Pfaundler syndromes, oral manifestations, 1233
Fluothane: new nonexplosive volatile anesthetic agent, 640 (Abst.)
Focus of infection, evaluation of method to determine presence of, 1130
Follicular sac, human, fate in heterologous transplantation, 308
Foramen, mental, radiodontic study, 127 (Abst.)
Fracture(s), compound comminuted, of mandible, 1469
jaw, delayed treatment, 1408 (Abst.)
of maxillary tuberosity occurring during tooth extraction, 409
osteomyelitis in line of, reconstruction of mandible following bone loss due to, 663
progressive atrophy of mandible complicated by, 4
root, 1485
management, 1475
styloid, diagnosis and treatment, 1423
treatment, 640 (Abst.)

G

Gamma globulin in treatment of recurrent herpetic gingivostomatitis, 165
Gastrointestinal tract and oral mucosa, multiple malignant tumors involving, 295
Georgetown University, 509, 638, 1405
Giant-cell epulis, hyperparathyroidism first suspected through, 482
granuloma, peripheral, 974
reparative granuloma, 383 (Abst.)
peripheral, 805
Gingiva(e), 802
attached, solitary xanthoma, 449
mandibular, squamous-cell carcinoma arising in, results of treatment, 252 (Abst.)
normal and inflamed, histochemical study of cytochrome oxidase in, 1501
Gingival changes observed in arteriosclerotic men, 1181
mucoperiosteal flaps, healing of, 89
and tooth injury in young adult male rats, response of periodontium, pulp, and salivary glands, 613, 734, 870
Gingivitis, 802
caused by *Histoplasma capsulatum*, 251 (Abst.)
chronic desquamative, 1300
during pregnancy, 804
Gingivostomatitis, recurrent herpetic, treated with gamma globulin, 165
Globulomaxillary cyst, ameloblastoma arising within, 1352
Glomera as vascular components in periodontal membrane; studies on etiology of periodontosis, 157
Glossectomy, partial, rehabilitation after, 1149 (Abst.)
Glucose concentrations in parotid fluid and blood serum following intravenous glucose loading, 1010
-tolerance pattern, normal, 819
Gly-Oxide; investigations of new dental chemotherapeutic agent in presence of blood, 678
Gonadal dose in children during intraoral radiography, measurement, 1081
Grand Central Hospital (New York City), internships and residency, 1146, 1401, 1507
Granuloma, giant cell reparative, 383 (Abst.)
peripheral giant-cell, 974
reparative, 805
pyogenicum, 809
Greater Washington Periodontal Society, 246

H

Hansen, Captain Louis S., new chief at Dental and Oral Pathology Division, Armed Forces Institute of Pathology, 1460
Head and neck, benign and malignant neurilemmomas, 1407 (Abst.)
Headache caused by bruxism, 639 (Abst.)
of dental origin, 1314
Heart disease, arteriosclerotic, gingival changes observed in men with, 1181
Heat and tobacco tar, experimental production of cancer with, 1513 (Abst.)
Hemangioendothelioma, infantile, of tongue, 320

Hematoma following dental extractions in patient receiving unreported anticoagulant therapy, 791
Hemophilia(s) and pseudohemophilias, some thoughts on, 639 (Abst.)
tooth extraction in patients with, 1167
Hemorrhage, postoperative, due to deficiency of plasma thromboplastin antecedent and plasma thromboplastin component, 671
Herpes labialis, recurrent, and recurrent aphthous ulcerations in professional school student population, 1191, 1317, 1438
simplex, chronic, vaccinotherapy, 1514 (Abst.)
zoster—primary manifestation of chronic lymphatic leukemia, 1429
Herpetic gingivostomatitis, recurrent, treated with gamma globulin, 165
Heterotransplants, parotid gland, and Shope papilloma virus, 598
Histologic appearance and sodium and potassium content of rat submaxillosublingual glands, effect of pilocarpine stimulation, 240
changes produced by pressure injection of whole blood into intermedullary spaces of dog mandibles, 605
Histoplasma capsulatum as cause of gingivitis, 251 (Abst.)
Hodgkin's disease treated with nitrogen mustard and radiation, complicated by osteomyelitis of mandible, 651
Horizontal angulation in maxillary cuspid roentgenography, 710
Humboldt University of Berlin Medical Faculty (Charité), 762
Hurler-Pfaundler, Fitzgerald-Gardner, Pringle-Bourneville, Robin, and adrenogenital syndromes, oral manifestations, 1233
Hydrogenated fat–zinc bacitracin periodontal dressing, 494
Hypercortisonism, osteoporosis of jaws associated with, 1445
Hyperparathyroidism first suspected through giant-cell epulis, 482
primary, 1408 (Abst.)
Hyperplasia, unilateral, and exostosis of mandibular condyle, 387
Hypnosis as adjunct in anesthesiology, 1513 (Abst.)
Hypochlorite for control of bleeding, 1026

I

Illinois section of American Dental Society of Anesthesiology, 1506
Ilosone, propionyl ester of erythromycin, 539
Implant(s); implant dentures—indications and procedures, 125 (B. rev.)
silver, in situ fifty-one years after resection of mandible, 384 (Abst.)
subperiosteal Vitallium, in dogs, 1153
Incisor, mandibular permanent, dilaceration, 1249
Indonesian Dental Association, 121
Infancy, syndrome of acute osteomyelitis of superior maxilla in early, 127 (Abst.)
Infantile hemangioendothelioma of tongue, 320
Infected deciduous molars, bacteriologic study, 1382
Infection and edema, buccal streptokinase in treatment, 812
focus of, evaluation of method to determine presence of, 1130
Injuries, anterior cranial and upper facial, management, 383 (Abst.)
facial, surgical treatment, 764 (B. rev.)
radiation, surgery for, 1150 (Abst.)
Innervation of predentine, significance, 743
International Conference on Endodontics, second, summary of reports, 990
Intestinal and oral bacteria, effects of prolonged Antarctic isolation on, 117
polyposis, familial, with melanosis oris: syndrome of Peutz and Jeghers, 924
Intramedullary pin, surgically introduced, in mandible, roentgenographic follow-up, 1229
Intraoral cancer, elective neck dissection for, 1150 (Abst.)
carcinoma, end results and causes of failure in treatment, 1408 (Abst.)
cavity, leukoplakia, 895 (Abst.)
leukoplakia and associated carcinoma, comparisons of histologic, cytologic, and clinical findings, 412
radiography, measurement of gonadal dose in children during, 1081
Intrathoracic suppuration and dental sepsis, 895 (Abst.)
Intubation, endotracheal, endotracheal anesthesia and trauma incident to, 543

J

Jaw(s) fracture treatment, delayed, 1408 (Abst.)
multiple hereditary familial epithelial cysts with associated anomaly of trichoepithelioma, 111
myxoma, 1462
osteoporosis associated with hypercortisonism, 1445
upper, resection and immediate restoration, 1033
Jewish Chronic Disease Hospital (Brooklyn), 638

K

Kenalog; triamcinolone acetonide, 937
Keratoacanthoma of oral cavity, 844

L

Lactobacillus acidophilus; effects of prolonged Antarctic isolation on oral and intestinal bacteria, 117

Lesions, oral, acute and chronic, new oral surface anesthetic for, 630
and associated, experimental use of proteolytic enzymes in treatment, 641
Leukemia, chronic lymphatic, herpes zoster as primary manifestation, 1429
Leukoplakia of bladder, 894 (Abst.)
buccalis and related lesions, treatment with estrogenic hormone, 1513 (Abst.)
intraoral, and associated carcinoma, comparisons of histologic, cytologic, and clinical findings, 412
of intraoral cavity, 895 (Abst.)
Lip, double, 236, 627
Lipoid proteinosis; unusual stomadromes, 257
Lipoma of oral cavity, 767
Low denture-tolerance syndrome; treatment of precancerous oral mucous membranes, 1065
Loyola University, 1148
Lung abscess, chronic, and bronchiectasis from aspiration of dental bridge, 1023
oral benign lesion progressing to epidermoid carcinoma with metastasis to, 977

M

Macroglobulinemia of Waldenström, oral manifestations, 104
Malignant melanoma of oral cavity, 584
primary in oral cavity, 971
tumors, multiple, involving oral mucosa and gastrointestinal tract, 295
Mandible, ameloblastoma, 648
benign neoplastic fibro-osseous lesion, 716
cervicofacial actinomycosis, 775
compound comminuted fracture, 1469
dog, histologic changes produced by pressure injection of whole blood into intermedullary spaces, 605
edentulous, osteosclerosis simulating carious unerupted cuspid, 668
mixed tumor of salivary glands, 519
neurofibromatosis (Von Recklinghausen's disease) with involvement of, 76
neurogenic fibrosarcoma in, observed about one month after extraction of lower third molar, 203
odontogenic cyst, 523
ossifying fibroma, 1288
osteoma, 1356
osteomyelitis of, Hodgkin's disease treated with nitrogen mustard and radiation complicated by, 651
progressive atrophy complicated by fractures, 4
reconstruction following bone loss due to osteomyelitis in line of fracture, 663
roentgenographic follow-up of surgically introduced intramedullary pin in, 1229
solitary myeloma, 23
Mandibular canal enlargement, 1350 (Roentgeno-Oddities)
bilateral, 837 (Roentgeno-Oddities)
Mandibular—Cont'd
condyle, exostosis of, and unilateral hyperplasia, 387
joint, chronic unilateral dislocation treated surgically by high condylectomy, 12
dysfunction, diagnosis, 1106
odontectomy, "dry socket" after, and use of soluble tetracycline hydrochloride, 38
Massachusetts Memorial Hospitals (*See* Boston University)
Maxilla, breast carcinoma metastatic to, 1099
myxoma of left, 139
odontoma, 513
superior, syndrome of acute osteomyelitis in early infancy, 127 (Abst.)
Maxillary antrum, dentigerous cyst involving supernumerary maxillary molar with encroachment on, 401
cuspid roentgenography, horizontal angulation, 710
sinus abnormalities, 447 (Roentgeno-Oddities)
olfactory neuroblastoma (esthesioneuroepithelioma), 908
tuberosity, fractures of, occurring during tooth extraction, 409
Maxillofacial prosthesis, temporary, in surgical—oncologic cases, 33
Medical College of Virginia, 248, 1279, 1509
Medication, preanesthetic, 639 (Abst.)
for children, 127 (Abst.)
Medicine (*See* Oral Medicine)
Melanoma, malignant, of oral cavity, 584
primary in oral cavity, 971
Melanosis oris, familial intestinal polyposis with: syndrome of Peutz and Jeghers, 924
Mental foramen, radiodontic study, 127 (Abst.)
Meprobamate and promazine hydrochloride, dental use for mentally retarded and emotionally disturbed patients, 423
Mercurial necrosis of cheek, 54
Methohexital sodium, 1330
anesthesia for ambulant dental patient, 1072
for pediatric anesthesia in difficult and handicapped patients, 686
Mid-Atlantic States Conference on Dentistry, eleventh, 377
Mid-Continent Dental Congress, 891, 1020
Mikulicz's disease, 1387
Mineralized dental tissues, 70
Mixed tumor of salivary glands of mandible, 519
Molar(s), infected deciduous, bacteriologic study, 1382
lower first permanent, retention, 1425
third, neurogenic fibrosarcoma in mandible observed about one month after extraction of, 203
rat, histologic effects of various drugs on amputated pulps, 455

Molars—Cont'd
supernumerary maxillary, dentigerous cyst involving, with encroachment on maxillary antrum, 401
Moniliasis, cutaneous and mucous membrane, 128 (Abst.)
Morrisania City Hospital, surgical internship, 1147
Mount Sinai Hospital of New York City, 1401
accepting internship and residency applications, 1402
oral pathology conference, 1278
Oral Pathology Laboratory, 123, 378, 637
Mouse, germ-free, and conventional control; studies on periodontal disease in mouse, 1134
Mouth cancer, early, exfoliative cytology for detection, 994
edentulous, delayed treatment of unerupted teeth in, 918
squamous-cell carcinoma of floor of, treatment, 253 (Abst.)
Mucoperiosteal flaps, gingival, healing of, 89
Mucous membranes, familial white folded dysplasia of, 252 (Abst.)
oral, color shade chart for study of, 1061
precancerous, treatment, 1065
triamcinolone acetonide in treatment of acute and chronic lesions, 170
Myeloma, solitary, of mandible, 23
Myxoma of jaws, 1462
of left maxilla, 139

N

Narcosedation, synergistic basal, 176
Neck dissection, elective, for intraoral cancer, 1150 (Abst.)
radical for cervical lymph node metastasis of intraoral carcinoma, 1514 (Abst.)
and head, benign and malignant neurilemmomas, 1407 (Abst.)
upper, and parotid gland, vascular abnormalities about, 384 (Abst.)
Necrosis, mercurial, of cheek, 54
Neomycin-bacitracin, root canal treatment with, as cause of flare-up of allergic eczema, 589
Neoplastic fibro-osseous lesion, benign, of mandible, 716
Nerve, seventh, spontaneous regeneration, 1513 (Abst.)
Neurilemmomas, benign and malignant, of head and neck, 1407 (Abst.)
large sublingual, unusual occurrence, 1163
Neuroblastoma, olfactory, of maxillary sinus, 908
Neurofibromatosis (Von Recklinghausen's disease) with involvement of mandible, 76
Neurogenic fibrosarcoma in mandible observed about one month after extraction of lower third molar, 203
Neuzeitliche kieferorthopädische praxis, 1022 (B. rev.)
Nevus, basal-cell, 252 (Abst.)
pregnancy tumor superimposed upon, 657
New York Institute of Clinical Oral Pathology, Inc., 637, 1145
New York State Dental Society of Anesthesiology, 1145
New York University, 250 1146
Nitrous oxide in dentistry—dangers and alternatives, 1510 (B. rev.)
Northwestern University, 123

O

Occlusal equilibration and temporomandibular joint dysfunction, 125 (B. rev.)
Odontectomy, mandibular, "dry socket" after, and use of soluble tetracycline hydrochloride, 38
Odontogenic cyst of mandible, 523
Odontoma of maxilla, 513
Ohio Dental Society of Anesthesiology, 1277
Ohio State University, 1147
Olfactory neuroblastoma (esthesioneuroepithelioma) of maxillary sinus, 908
Oncologic-surgical cases, use of temporary maxillofacial prosthesis in, 33
Open-bite, anterior, or retrognathia corrected by vertical sliding osteotomy of ramus, with or without bone graft, 1409
Operative procedures, symposium on pulp reaction to, 329
Oral actinomycetes, 384 (Abst.)
anatomy, 764 (B. rev.)
and histology, 743
benign lesion progressing to epidermoid carcinoma with metastasis to lung, 977
cancer, failures in treatment, 1408 (Abst.)
relationship to generalized connective tissue changes, 838
cavity, early cancer of, with special reference to cancer of tongue, 895 (Abst.)
keratoacanthoma, 844
lipoma, 767
malignant melanoma, 584
primary in, 971
surgery of, and technique of controlled tooth division, 511 (B. rev.)
change in Sturge-Weber's disease, 795
diagnosis of systemic disease, 896 (Abst.)
histology and embryology, Noyes', 1511 (B. rev.)
and intestinal bacteria, effects of prolonged Antarctic isolation on, 117
lesions, acute and chronic, new oral surface anesthetic for, 630
manifestations of Crouzon's disease, 1245
of Fitzgerald-Gardner, Pringle-Bourneville, Robin, adrenogenital, and Hurler-Pfaundler syndromes, 1233
medicine, 54, 165, 412, 534, 671, 795, 924, 1061, 1191, 1314, 1429
mucosa and gastrointestinal tract, multiple malignant tumors involving, 295

Oral—Cont'd
mucous membranes, acute and chronic lesions of, triamcinolone acetonide in treatment, 170
atlas of clinical pathology of, 1510 (B. rev.)
color shade chart for study of, 1061
precancerous, treatment, 1065
pathologic conditions, prenatal factors, 569
pathologist looks at practice of dentistry, 331
pathology, 70, 203, 449, 569, 716, 838, 971, 1099, 1233, 1249, 1352, 1460
Archives now available, 1147
roentgenographic findings, relationship of three-hour blood glucose to, 1447
roentgenology, 63, 188, 341, 432, 552, 696, 819, 953, 1081, 1218, 1343, 1445
smears in diagnosis of carcinoma and premalignant lesions, 897
surgery at crossroads, 129
and oral medicine, studies in diagnosis, 482, 994
operative, 4, 129, 387, 513, 641, 767, 897, 1023, 1153, 1283, 1409
quieting agent for anxiety in, 59
tetracycline with glucosamine (Cosa-Tetracyn) in, 530
textbook of, 124 (B. rev.)
surgical procedures, bacterial invasion of blood after, 1022 (Abst.)
teaching, chairside, patient's reaction, 913
treponemes, parenterally inoculated, studies on survival in guinea pig, 883
ulcerations, experimental production in rat, 1267
Oronasal fistula, plastic closure, 146
Orthodontia, textbook of, 125 (B. rev.)
Ossifying fibroma of mandible, 1288
Ostamer (polyurethane polymer), use in fractured and diseased bones, 896 (Abst.)
Osteitis, localized; study of dry sockets, 1046
Osteogenesis imperfecta, incremental pattern of dentine in case of, 70
Osteoma of mandible, 1356
Osteomyelitis, acute, of superior maxilla in early infancy, 127 (Abst.)
in line of fracture, reconstruction of mandible following bone loss due to, 663
of mandible, Hodgkin's disease treated with nitrogen mustard and radiation complicated by, 651
Osteoporosis, alveolar, and pulpal death associated with compulsive bruxism, 1371
of jaws associated with hypercortisonism, 1445
Osteosclerosis in edentulous mandible simulating carious unerupted cuspid, 668
Osteotomy, vertical sliding, of ramus, with or without bone graft, for correction of anterior open-bite or retrognathia, 1409

P

Pachyonychia congenita; unusual stomadromes, 257
Pacific Coast Workshop, 761
Pain, facial, 1512 (Abst.)
sensitivity of dentine and pulp, topical use of corticosteroids for relief, 594
Palatopharyngeal mechanism, roentgenography of, use of dental x-rays in, 1085
Papillary cystadenoma of minor salivary gland origin, 452
Parotid fluid and blood serum, glucose concentrations following intravenous glucose loading, 1010
flow rate, 1488
as medium for determination of human adrenocortical status, 878
gland extract, effect on hard and soft tissues of growing mice, 875
flow rate and parotid fluid urea concentration, 1013
heterotransplants and Shope papilloma virus, 598
tumors, 639 (Abst.)
and upper neck, vascular abnormalities about, 384 (Abst.)
Pathologic conditions, oral, prenatal factors, 569
Pathologist, oral, looks at practice of dentistry, 331
Pathology (*See also* Oral Pathology)
clinical, of oral mucous membrane, atlas, 1510 (B. rev.)
periapical, conservative treatment of extensive areas of, 858
Pediatric anesthesia in difficult and handicapped patients, new intravenous barbiturate, 686
Periadenitis mucosa necrotica recurrens, 750
Periapical pathology, conservative treatment of extensive areas of, 858
Periodontal disease in mouse, 1134
dressing, hydrogenated fat–zinc bacitracin, 494
therapy, 1280 (B. rev.)
tissues of Syrian hamster, effects of desalivation, 470
widening; relationship of three-hour blood glucose to oral roentgenographic findings, 1447
Periodontally and endodontically involved tooth, therapy, 361
Periodontics, 43, 157, 1051, 1181, 1300
Periodontium, pulp, and salivary glands, response to gingival and tooth injury in young adult male rats, 613, 734, 870
Periodontosis, etiology, 157
Peripheral giant-cell granuloma, 974
reparative, 805
Permeability of root dentine, effects of endodontic treatments, 208
Peutz-Jeghers syndrome: familial intestinal polyposis with melanosis oris, 924

Pharmacology and therapeutics, 59, 170, 423, 539, 678, 812, 937, 1214
Physiologic response to dental stress, 368
Pilocarpine stimulation, effect on histologic appearance and sodium and potassium content of rat submaxillo-sublingual glands, 240
Pin, surgically introduced intramedullary, in mandible, roentgenographic follow-up, 1229
Pinhole camera, radiation protection via, 953
Plasma thromboplastin antecedent and plasma thromboplastin component, bleeding due to deficiency of, 671
Plastic closure of oronasal fistula, 146
Pleuropneumonia-like organisms, isolation and cultivation of filterable form resembling, from human dental pulp, 756
Polyposis, familial intestinal, with melanosis oris: syndrome of Peutz and Jeghers, 924
Polyurethane polymer (Ostamer), use in fractured and diseased bones, 896 (Abst.)
Potassium and sodium content and histologic appearance of rat submaxillo-sublingual glands, effect of pilocarpine stimulation, 240
Preanesthetic medication, 639 (Abst.)
for children, 127 (Abst.)
Precancerous lesion, problem of, 894 (Abst.)
oral mucous membranes, treatment, 1065
Predentine, innervation, 743
Pregnancy, gingivitis during, 804
tumor superimposed upon nevus, 657
Premalignant lesions, oral smears in diagnosis of carcinoma and, 897
Premolars, supernumerary mandibular, recurring, 964
Prenatal factors in oral pathologic conditions, 569
Pressure injection of whole blood into intermedullary spaces of dog mandibles, histologic changes produced by, 605
Primary amyloidosis of tongue, 534
Pringle-Bourneville, Fitzgerald-Gardner, Robin, adrenogenital, and Hurler-Pfaundler syndromes, oral manifestations, 1233
Profile-intensifying shield for cephalometric roentgenography, 1089
Promazine hydrochloride and meprobamate, dental use for mentally retarded and emotionally disturbed patients, 423
Prosthesis, removable partial denture, essentials, 126 (B. rev.)
temporary maxillofacial, in surgical-oncologic cases, 33
Proteinosis, lipoid; unusual stomadromes, 257
Proteolytic enzymes, experimental use in treatment of oral and associated lesions, 641
Pruritus ani, antibiotic-induced, treated with amino-acid ointment, 896 (Abst.)
Public Health Service (*See* Armed Forces and Public Health Service)
Pulp(s), amputated, of rat molars, histologic effects of various drugs, 455
dental, human, isolation and cultivation of filterable form resembling pleuropneumonia-like organisms from, 756
respiration of, effect of local anesthetics, 229
and dentine, pain sensitivity of, topical use of corticosteroids for relief, 594
healing, histochemical study, 868
periodontium, and salivary glands, response to gingival and tooth injury in young adult male rats, 613, 734, 870
reaction to operative procedures, symposium, 329
Pulpal death and alveolar osteoporosis associated with compulsive bruxism, 1371
Pulpitis, painful, 1360
Pulpless tooth, vibration of; evaluation of method to determine presence of focus of infection, 1130

R

Radiation dosage in dental offices, 696
injury, surgery for, 1150 (Abst.)
protection via pinhole camera, 953
reduction, direct, in dentistry, factors contributing to, 432
Radiography, intraoral, measurement of gonadal dose in children during, 1081
Radiotherapy of oral cancer, exfoliative cytology in, 1512 (Abst.)
Ramus, vertical sliding osteotomy of, with or without bone graft, for correction of anterior open-bite or retrognathia, 1409
Reese, Michael, Hospital (Chicago), 510, 1277, 1402
Regeneration, spontaneous, of seventh nerve, 1513 (Abst.)
Reiter's syndrome; unusual stomadromes, 257
Reparative granuloma, giant cell, 383 (Abst.)
peripheral, 805
Research, 89, 224, 470, 598, 734, 870, 1267
Resection of mandible, silver implant in situ fifty-one years after, 384 (Abst.)
of upper jaw and its immediate restoration, 1033
Resorption, idiopathic internal, of teeth, 723
internal, familial, 201 (Roentgeno-Oddities)
of retained teeth, 1097 (Roentgeno-Oddities)
Respiration of dental pulp, effect of local anesthetics, 229
Restorative materials, roentgenographic appearance, 68 (Roentgeno-Oddities)
Retained teeth, resorption, 1097 (Roentgeno-Oddities)
Retention of lower first permanent molar, 1425

Retrognathia or anterior open-bite corrected by vertical sliding osteotomy of ramus, with or without bone graft, 1409
Right-angle roentgenography; roentgenologic rebuttal, 1218
Robin, Fitzgerald-Gardner, Pringle-Bourneville, adrenogenital, and Hurler-Pfaundler syndromes, oral manifestations, 1233
Roentgenograms, periodic, are essential, 1232 (Roentgeno-Oddities)
temporomandibular joint, interpretation, 341
Roentgenographic findings in endentulous area, 1343
follow-up of surgically introduced intramedullary pin in mandible, 1229
localization of unerupted teeth, 439
Roentgenography, cephalometric, development of profile-intensifying shield, 1089
of enamel, 188
maxillary cuspid, horizontal angulation, 710
of palatopharyngeal mechanism with dental x-rays, 1085
right-angle; roentgenologic rebuttal, 1218
Roentgenologic rebuttal, 1218
Roentgenology (*See also* Oral Roentgenology)
dental, 125 (B. rev.)
workshop, eighth annual, 762
Roentgeno-Oddities, 68, 201, 351, 447, 567, 714, 837, 968, 1097, 1232, 1350, 1458
Roentgeno-Questions, 67, 199, 566, 713, 836, 967, 1096, 1231, 1349, 1457
Root apices of maxillary and mandibular posterior teeth, stereomicroscopic study, 728
canal treatment with bacitracin-neomycin as cause of flare-up of allergic eczema, 589
dentine, permeability of, effects of endodontic treatments, 208
fracture, 1485
management, 1475
resection, choice of anti-inflammatory agents, 396

S

Salicylamide derivative, evaluation of analgesic properties in comparison with placebo and aspirin, 1214
Saliva, bibliography on, 1511 (B. rev.)
Salivary gland(s) of mandible, mixed tumor, 519
minor, origin of papillary cystadenoma, 452
periodontium, and pulp, response to gingival and tooth injury in young adult male rats, 613, 734, 870
sialolithiasis of minor, 578
tumors, 850
Science, American men of, 382 (B. rev.)
Sclerosing solution in treatment of ameloblastoma, 151
Sealing properties of temporary filling materials, effect of temperature change, 982
Sepsis, dental, and intrathoracic suppuration, 895 (Abst.)
Shield, profile-intensifying, for cephalometric roentgenography, 1089
Shope papilloma virus and parotid gland heterotransplants, 598
Sialadenitis, chronic obstructive, of sublingual gland, 1295
Sialolithiasis of minor salivary gland, 578
Silver implant in situ fifty-one years after resection of mandible, 384 (Abst.)
Sinus, maxillary, olfactory neuroblastoma (esthesioneuroepithelioma), 908
Smith, Rowe, Memorial Foundation, 1401
Sodium and potassium content and histologic appearance of rat submaxillo-sublingual glands, effect of pilocarpine stimulation, 240
Solitary myeloma of mandible, 23
xanthoma of attached gingiva, 449
Southern California Academy of Endodontists, 891, 1020, 1145
Southern California Academy of Oral Pathology, 892, 1020
Southwest Society of Periodontists, 377
Squamous-cell carcinoma of floor of mouth, treatment, 253 (Abst.)
of mandibular gingiva, effects of treatment, 252 (Abst.)
Stensen's duct, correction of fistula, 382 (Abst.)
Sterilization of carious dentine, effectiveness of certain drugs for, 80
Stereomicroscopic study of 700 root apices of maxillary and mandibular posterior teeth, 728
Stomadromes, unusual—pachyonychia congenita, lipoid proteinosis, Reiter's syndrome, epidermolysis bullosa, Bonnevie-Ullrich-Turner's syndrome, and multiple hereditary telangiectasis, 257
Stomatitis, bismuth and uremic, 802
Streptokinase, buccal, in treatment of infection and edema, 812
Stress, dental, physiologic response, 368
response of systolic and diastolic blood pressures, 499
Sturge-Weber's disease, oral change, 795
Styloid fracture, diagnosis and treatment, 1423
Sublingual gland, chronic obstructive sialadenitis, 1295
neurilemmoma, large, unusual occurrence, 1163
Submaxillary gland(s), recurrent adenoid cystic carcinoma, 1103
response of periodontium, pulp, and salivary glands to gingival and tooth injury in young adult male rats, 870

Submaxillo-sublingual glands of rat, effect pilocarpine stimulation on histologic appearance and sodium and potassium content, 240
Subperiosteal Vitallium implants in dogs, 1153
Supernumerary mandibular premolars, recurcuring, 964
maxillary molar, dentigerous cyst involving, with encroachment on maxillary antrum, 401
Suppuration, intrathoracic, and dental sepsis, 895 (Abst.)
Surgery (*See also* Oral Surgery)
of oral cavity and technique of controlled tooth division, 511 (B. rev.)
for radiation injury, 1150 (Abst.)
Surgical anatomy and physiology, 1203
-oncologic cases, use of temporary maxillofacial prosthesis in, 33
Synergistic basal narcosedation, 176
Systemic disease, oral diagnosis, 896 (Abst.)

T

Teaching, chairside oral surgical, patient's reaction, 913
Teeth, idiopathic internal resorption, 723
maxillary and mandibular posterior, stereomicroscopic study of 700 root apices, 728
unerupted, in edentulous mouth, delayed treatment, 918
roentgenographic localization, 439
Telangiectasia, multiple hereditary; unusual stomadromes, 257
Temperature change, effect on sealing properties of temporary filling materials, 982
Temple University, 249, 763, 1146, 1278, 1508
Temporomandibular joint arthrosis, role of trauma in pathogenesis, 1514 (Abst.)
disorders, 126 (B. rev.)
dysfunction and occlusal equilibration, 125 (B. rev.)
roentgenograms, interpretation, 341
structure, 1203
Tetracycline with glucosamine (Cosa-Tetracyn) in oral surgery, 530
hydrochloride, soluble, for "dry socket" after mandibular odontectomy, 38
Tobacco tar and heat, experimental production of cancer with, 1513 (Abst.)
Tongue, black hairy, biocenologic approach with new concepts of treatment, 384 (Abst.)
cancer, early cancer of oral cavity with special reference to, 895 (Abst.)
carcinoma, treatment, 254 (Abst.)
congenital epidermoid cyst of anterior half, 1283
glandular tumors, clinicopathologic study, 383 (Abst.)
infantile hemangioendothelioma, 320
primary amyloidosis, 534
Tongue-tie; partial ankyloglossia, 787
Tooth division, controlled, technique of, and surgery of oral cavity, 511 (B. rev.)
endodontically and periodontally involved, therapy, 361
extraction, fractures of maxillary tuberosity occurring during, 409
in patients with hemophilia, 1167
and gingival injury in young adult male rats, response of periodontium, pulp, and salivary glands, 613, 734, 870
movement, minor, in general practice, 1511 (B. rev.)
Transplantation, heterologous, fate of human follicular sac in, 308
of tissues, 124 (B. rev.)
Trauma incident to endotracheal intubation, 543
role in pathogenesis of temporomandibular joint arthrosis, 1514 (Abst.)
Treatment, conservative, of extensive areas of periapical pathology, 858
delayed, of unerupted teeth in edentulous mouth, 918
planning in practice of dentistry, 126 (B. rev.)
Treponemes, oral, parenterally inoculated, survival in guinea pig, 883
Triamcinolone acetonide, 937
in treatment of acute and chronic lesions of oral mucous membranes, 170
Trichoepithelioma, multiple hereditary familial epithelial cysts of jaws with associated anomaly of, 111
Trypsin; experimental use of proteolytic enzymes in treatment of oral and associated lesions, 641
Tuberosity, maxillary, fractures of, occurring during tooth extraction, 409
Tufts University, 508, 893, 1021, 1405, 1508
announces eleventh Berkshire Conference in Periodontology and Oral Pathology, 379
Tumor(s), glandular, of tongue, clinicopathologic study, 383 (Abst.)
malignant, of maxilla; resection of upper jaw and its immediate restoration, 1033
mixed, of salivary glands of mandible, 519
multiple malignant, involving oral mucosa and gastrointestinal tract, 295
of parotid glands, 639 (Abst.)
pregnancy, superimposed upon nevus, 657
of salivary glands, 850

U

Ulcerations, oral, experimental production in rat, 1267
recurrent aphthous, and recurrent herpes labialis in professional school student population, 1191, 1317, 1438
Ultrasonic instrument, curettage by, 43
Unerupted cuspid, carious, osteosclerosis in edentulous mandible simulating, 668

Unerupted—Cont'd
teeth in edentulous mouth, delayed treatment, 918
roentgenographic localization, 439
University of Alabama, 122, 247, 1019, 1147, 1276, 1402
of California, 1148
of Illinois, 123, 249, 250, 378, 1146, 1404
of Minnesota, 508
of Mississippi, 893
of North Carolina, 638
of Oregon, 378
of Pennsylvania, 249, 378, 638, 1277, 1404, 1507
research and teaching traineeships, 378, 1404, 1507
of Pittsburgh, 1404
of Washington, 510, 1509
graduate program in dentistry, 508
Urea concentration of parotid fluid and parotid gland flow rate, 1013
Uremic stomatitis, 802

V

Vaccinotherapy of chronic herpes simplex, 1514 (Abst.)
Varidase, buccal, in treatment of infection and edema, 812
Vascular abnormalities about parotid gland and upper neck, 384 (Abst.)
Veterans Administration Hospital in Philadelphia, internships available, 1279
Vibration of pulpless tooth; evaluation of method to determine presence of focus of infection, 1130
Virus, Shope papilloma, and parotid gland heterotransplants, 598
Vitallium implants, subperiosteal, in dogs, 1153
Von Recklinghausen's disease with involvement of mandible, 76

W

Waldenström, macroglobulinemia of, oral manifestations, 104
Western Reserve University, 379

X

Xanthoma, solitary, of attached gingiva, 449
X-ray(s), dental, use in roentgenography of palatopharyngeal mechanism, 1085
equipment, dental, of future, 194
machine, 65 or 90 kilovolt? 552
Xylocaine, 894 (B. rev.)

Y

Yeshiva University, Albert Einstein College of Medicine, 1279, 1406

Editorials

Composing a Scientific Article, 1
Dental Radiology—Specialty Status, 1151
Legal Responsibility, 1281
The Oral Pathologist—a Specialist, 255
The Place of Endodontics in Dental Practice, 385

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TABLE OF CONTENTS ON PAGE ONE

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Contents for December, 1960

OPERATIVE ORAL SURGERY

Anterior Open-Bite or Retrognathia Corrected by a Vertical Sliding Osteotomy of the Ramus, With or Without Bone Graft: An Improved Technique. Robert W. Christensen, D.D.S., Pasadena, Calif. 1409

Retrognathia is a distressing and disfiguring condition. The present report describes the treatment of two cases.

Diagnosis and Treatment of a Styloid Fracture. Thomas A. Armao, D.D.S., Brooklyn, N. Y. 1423

Styloid fracture is an unusual injury, which should be recognized.

EXODONTIA

Retention of a Lower First Permanent Molar. Cornelia Terner, M.D., Debrecen, Hungary 1425

An unusual problem in exodontia.

ORAL MEDICINE

Herpes Zoster—A Primary Manifestation of Chronic Lymphatic Leukemia. Constantine P. Chaconas, D.D.S., Silver Spring, Md. 1429

This paper presents an unusual problem of diagnosis in an oral medical case.

Recurrent Aphthous Ulcerations and Recurrent Herpes Labialis in a Professional School Student Population. III. Oral Examinations. Irwin I. Ship, D.M.D., Alvin L. Morris, D.D.S., Ph.D., Roy T. Durocher, D.D.S., and Lester W. Burket, D.D.S., M.D., Bethesda, Md., and Philadelphia, Pa. 1438

Continuation of an interesting study.

ORAL ROENTGENOLOGY

Osteoporosis of the Jaws Associated With Hypercortisonism. Edward C. Stafne, D.D.S., and Stanley A. Lovestedt, D.D.S., Rochester, Minn. 1445

A case of hypercortisonism with osseous manifestations in the mandible is presented and discussed.

Relationship of Three-Hour Blood Glucose to Oral Roentgenographic Findings. L. Manson-Hing, D.M.D., and E. Cheraskin, M.D., D.M.D., Birmingham, Ala. 1447

Findings are presented which relate roentgenographic evidence of interdental bone loss to both hypo- and hyperglycemia.

Roentgeno-Questions 1457

Roentgeno-Oddities 1458

(Contents continued on Page Three)

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Contents continued from page one

ORAL PATHOLOGY

New Chief at Dental and Oral Pathology Division, Armed Forces Institute of Pathology – 1460

Myxoma of the Jaws. Nelson D. Large, D.D.S., Salisbury, N. C., and Harold H. Niebel, B.S., D.D.S., and Walter H. Fredricks, D.D.S., Chicago, Ill. – – – 1462

Myxoma of the jaws may be odontogenic or osteogenic in origin. This article presents two cases of the much rarer osteogenic type.

Compound Comminuted Fracture of the Mandible. Robert A. Atterbury, B.S., D.D.S., F.I.C.D., Oak Park, Ill. – – – – – – – – – – – – – – – – 1469

A case in which a difficult fracture was reduced by multiple methods is described.

ENDODONTICS

Management of Root Fracture. Lonnie C. Sibley, D.D.S., Marshall, Texas – – 1475

The rationale for conservative treatment of root fractures is discussed.

Root Fracture. F. James Marshall, D.M.D., M.S., Chicago, Ill. – – – – – – – 1485

A contribution to the treatment of tooth root fractures.

SECTION BY THE ARMED FORCES AND PUBLIC HEALTH SERVICE

Parotid Fluid Flow Rate. Ira L. Shannon, Major, USAF (DC), and John R. Prigmore, Captain, USAF (MSC) – – – – – – – – – – – – – – – – 1488

The composition of the parotid gland is of great interest to the dentist.

Histochemical Study of Cytochrome Oxidase in Normal and Inflamed Gingiva. M. S. Burstone, D.D.S., Bethesda, Md. – – – – – – – – – – – – – – – 1501

A striking increase in cytochrome oxidase activity was found in chronic gingivitis.

ANNOUNCEMENTS

Announcements – 1506

REVIEWS OF THE LITERATURE

Book Reviews – 1510

Abstracts of Current Literature – 1512

INDEX

Index – 1515

(Editorial and Business Communications on page 7)

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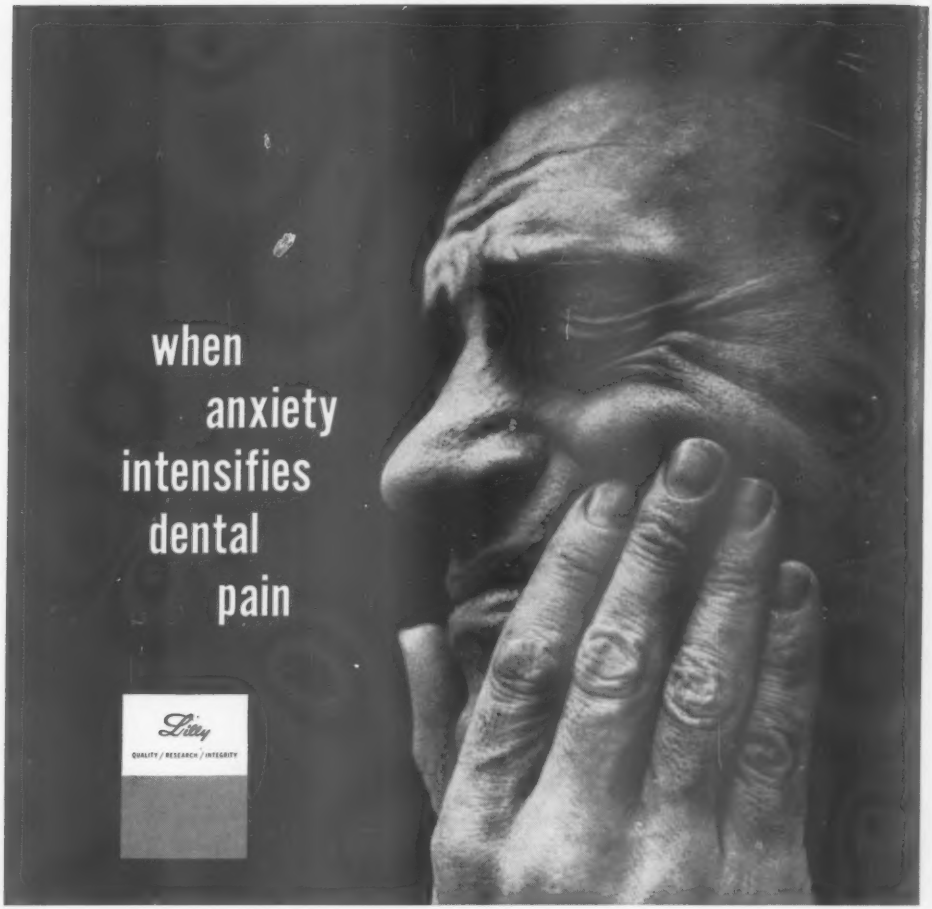

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Review of the literature
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CONTENTS

PART I—DESCRIPTIVE ANATOMY

1. The Skull
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3. The Temporomandibular Articulation
4. The Viscera of Head and Neck
5. The Blood Vessels of Head and Neck
6. The Lymphatic System of Head and Neck
7. The Nerves of Head and Neck

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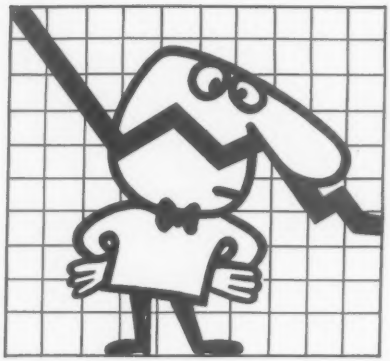

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New evidence
helps resolve
current controversy
over
oral penicillins

Source of confusion:

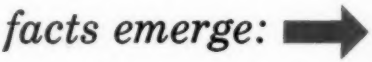
dependence on microgram "blood levels" of oral penicillins as the measure of therapeutic value

Concerning this, McCarthy and Finland[1] state:

> "Some [oral penicillins] may be well absorbed but of low activity, and others may be of higher activity and less well absorbed but nevertheless may provide more total activity."

To clarify the problem:

In their study, McCarthy and Finland compared the serum levels by weight and the serum antibacterial activity of several penicillins in a series of well-controlled crossover studies. Among the oral penicillins tested were penicillin V potassium and phenethicillin (potassium alpha-phenoxyethyl penicillin). The subjects used in the study were normal, healthy young men. Before and after ingestion of the antibiotics, the activity of the subjects' serums was tested against three common pathogens —hemolytic streptococcus 98, Type 3 pneumococcus, and *Staphylococcus aureus* 209P—by the twofold tube dilution method.

These facts emerge: 

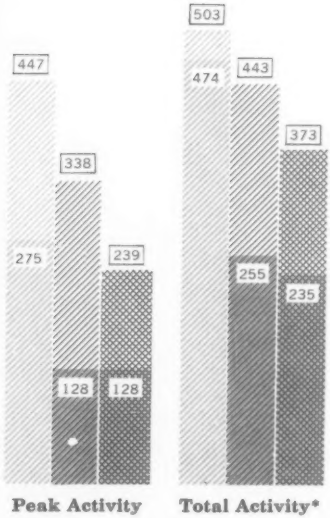
Although the concentrations of phenethicillin were higher, "penicillin V [potassium] provided greater activity than phenethicillin against the streptococcus and pneumococcus . . . [and] at least equivalent activity against the staphylococcus. . . ."

—McCarthy, C. G., and Finland, M.: Absorption and Excretion of Four Penicillins, New England J. Med., 263:315 (August 18), 1960.

Pot
Phe
Phe

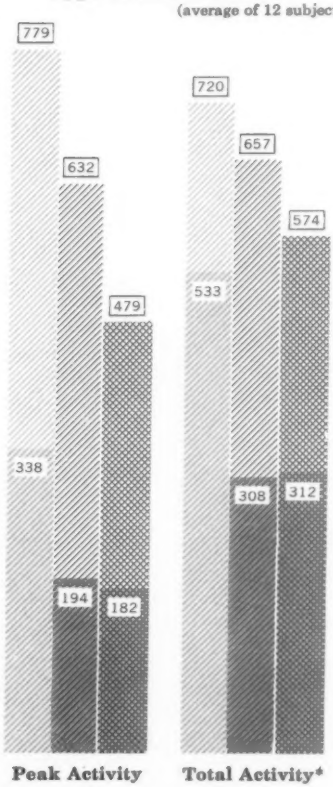
Lined ar
shaded ar

Number of times serum can be diluted and still inhibit **Streptococcus 98**[1] (average of 12 subjects)



Peak Activity **Total Activity***

Number of times serum can be diluted and still inhibit **Type 3 Pneumococcus**[1] (average of 12 subjects)



Peak Activity **Total Activity***

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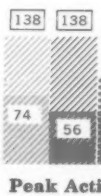


Peak Acti

*Shows "area under the curve," that is, the average of serum dilutions taken over a five-hour period.

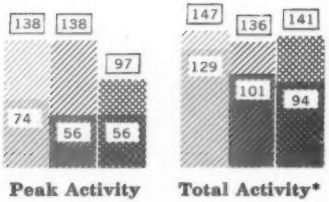
Potassium penicillin V

Phenethicillin (capsules)

Phenethicillin (tablets)

Lined areas represent fasting levels; shaded areas depict levels after food.

Number of times serum can be diluted and still inhibit **Staphylococcus 209P**[1]
(average of 12 subjects)



"Blood Levels" vs. "ABA"

McCarthy and Finland's conclusions extend and confirm their previous observations[2] and those of Griffith.[3] They demonstrate that phenethicillin gives higher "blood levels" in micrograms, but, in terms of antibacterial activity in the serum ("ABA"), potassium penicillin V is clearly superior against the common pathogens (streptococcus and pneumococcus) and at least equal against the staphylococcus.

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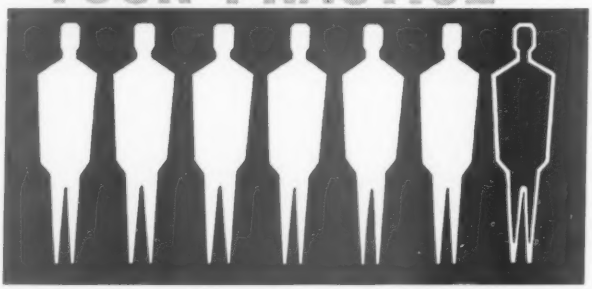

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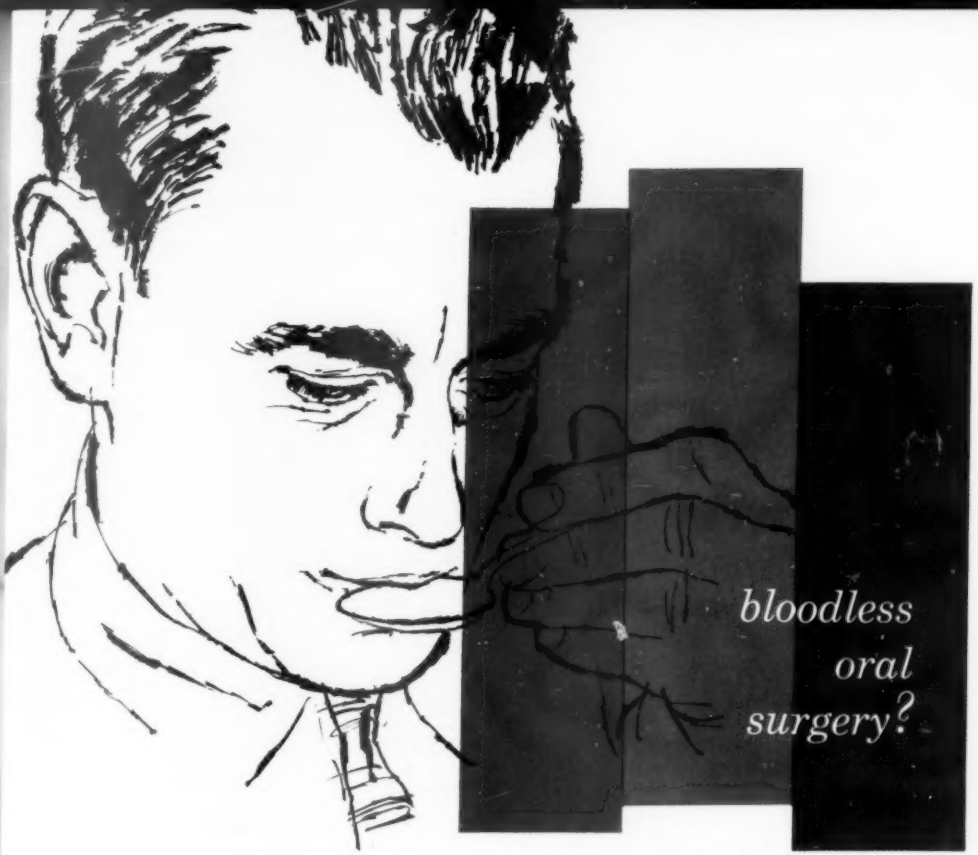

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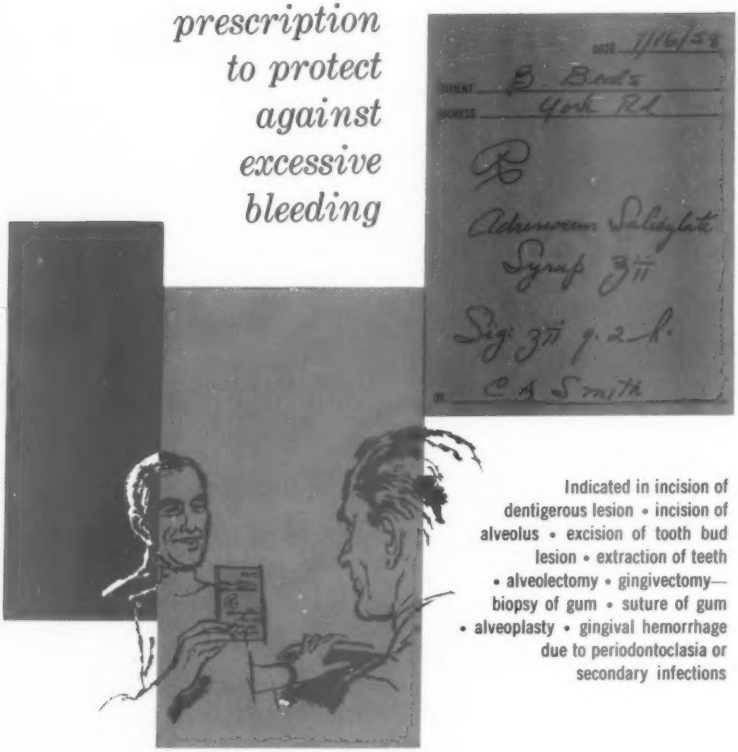

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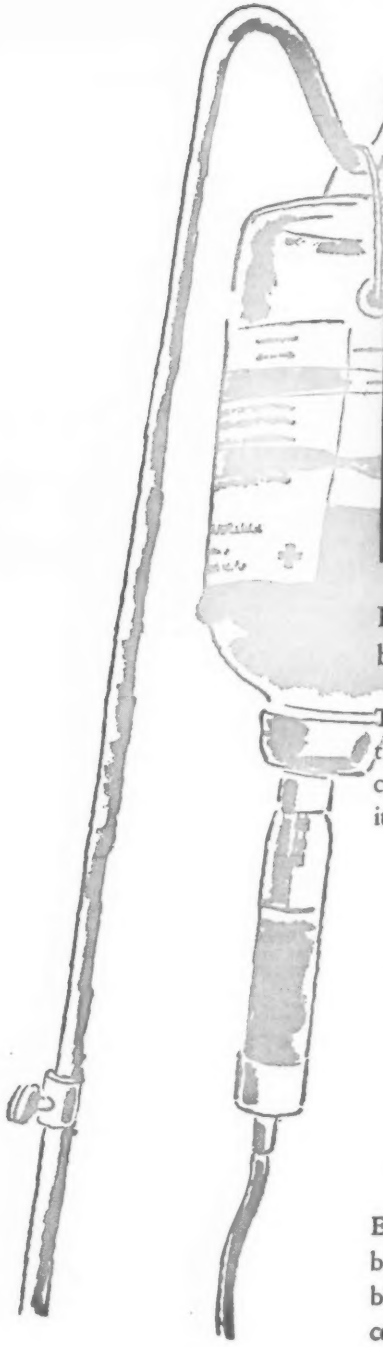

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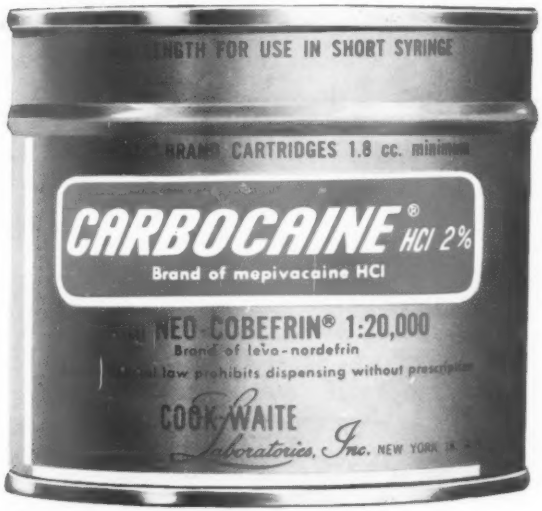

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- Carbocaine **frees patients** sensitized to procaine **from allergic responses** to procaine-related local anesthetics.
- Carbocaine's **effectiveness** is consistently **excellent.**
- Carbocaine's **onset** is very, very **rapid,** frequently reported as "immediate".

We urge **you** to **try Carbocaine now** and see for yourself. Your dental dealer stocks it in 1.8 cc. min. cartridges, 50 cartridges per can. Or write for clinical samples and detailed literature to the pioneer in dental local anesthesia...

9819

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